ARCHIVES OF OPHTHALMOLOGY.

ON THE PROBABLE HEMORRHAGIC ORIGIN OF THE STRIATED AFFECTIONS OF THE RETINA.[1]

With reports of cases from the practice of Dr. H. KNAPP and the author.

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(*With two illustrations.*)

ONE of the weak points in ophthalmoscopic diagnosis is the frequency with which we are unable to distinguish conditions that are the result of hemorrhage, from conditions that are due to primary inflammation; although the more careful study of retinal affections in late years has taught us that many conditions supposed to be inflammatory represent in fact the metamorphosis of hemorrhage.

Of the three distinct varieties of new-formed striations in the retina, two are known to be the result of hemorrhage, while the genesis of the third is still unknown.

A case to which I wish to ask your attention to-night combines in a measure the characteristics of all these varieties, and in it the development of striations from a fresh hemorrhage was clearly followed. This, together with the analogous appearance of many cases of the unexplained variety of retinal striations, leads me to suggest that this variety also may be the result of hemorrhage.

The first variety is the condition in which on or just beneath the surface of the retina, and mostly overlying the retinal vessels, are strands of bluish-white glistening membrane. This has frequently been seen to follow traumatic or idiopathic hemorrhage, and Manz has called it retinitis proliferans. This designation seems proper since it has recently been shown that there is a proliferation of Müller's

[1] Read before the Ophthalmological Section of the New York Academy of Medicine, November 19, 1894.

fibres and a deposition among them of new connective tissue.

The second variety is the very rare condition in which a number of narrow branched brown stripes lying *beneath* the vessels of the retina run from the periphery of the fundus toward the disc. Plange,[1] who described the first case reported, saw these stripes develop in the course of repeated hemorrhages. Knapp[2] reported a case calling the affection angioid streaks in the retina, and Stephenson[3] reported another in which many of the brown stripes were bordered on one side only by a broader white stripe.

The third variety is the rather rare condition in which lying beneath the retinal vessels are narrow yellowish-white striæ, sometimes branched, and often bordered on either side by a narrow line of pigment; the striæ extending in general direction from the periphery toward the disc. This affection was pictured by Jaeger and was later called by Nagel retinitis striata. Its etiology is unknown.

All of these varieties of striations are permanent conditions, and are not easily confounded with the inflammatory stripes in *syphilitic* retinitis, which run in the direction of the vessels and may disappear under specific treatment.

The case to be reported and most of those to be briefly mentioned later, have been seen in the private practice of Dr. Knapp, and through his kindness I am permitted to make use of them.

Miss M., aged forty-three, was first seen on July 5, 1894, when she complained of gradual failure of sight for a year. The patient was of slender build, was nervous and anæmic, and had frequent attacks of giddiness. She had had no serious illness of recent years, and there were no discoverable organic affections, and no menstrual disturbance. The acuteness of vision was R. $\frac{20}{200}$; L. $\frac{20}{100}$, not improved with glasses. The limits of the fields for white and colors were normal. The media were clear. The optic discs and the retinal vessels presented no peculiarities. About the disc in each eye was an irregularly outlined zone in which the

[1] ARCH. OF OPHTH., xxi., 2, p. 283, 1892.
[2] *Ibid.*, p. 299.
[3] *Trans. Ophth. Soc., Un. K.*, xii., 1892.

retina was somewhat opaque, obscuring the red reflex from the choroid. In each macular region there was disturbance of pigmentation with scattered small hemorrhages, dark spots, and superficial bluish-white membranous patches.

Radiating from the pale zone about the disc, in each eye, was a system of branched striations of dark-brown color, having each about the width of a vein on the optic disc, and lying beneath the retinal vessels. In the left eye this system of angioid streaks closely resembled those described in previous cases, and does not particularly interest us. In the right eye many of these

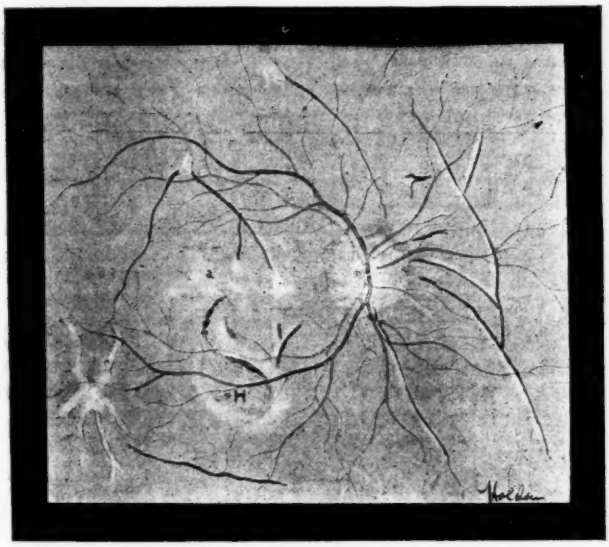


FIG. I.

Pigmented Angioid Streaks in the retina. With a hemorrhage, "× H" in the process of absorption and a new branched streak forming above it. *Erect image.*

angioid streaks were bordered on one side only, by a light streak double the width of the dark. In the temporal periphery of the right fundus was a patch of opacity in the retina, from which narrow processes branched out, each bordered with a delicate line of pigment. Temporally and below the disc a large hemorrhage of

bright red color, irregularly branched in form though sharply outlined, lay beneath a retinal vein. When seen three weeks later the hemorrhage had assumed a more regularly elliptical form, and the surrounding retina had become milky. The hemorrhage then began to grow paler below, while the superior margin remained bright red. As the hemorrhage became further absorbed a narrow process of red color pushed upward from its superior margin and gradually extended a distance of two disc-diameters in a curved streak, but slightly broader than a vein on the disc. It still was red when the patient was last seen, four months after the first visit.

While the hemorrhage was undergoing absorption from below, there appeared just above it a short streak of dark-brown color, bordered on one side by a broader light streak. This double streak branched like a Y, as is shown in the drawing, and one branch gradually extended upward until it became two disc-diameters in length, while the shorter branch became partially absorbed. This new streak was similar in color and conformation to the older ones.

When examined in the erect image all the dark streaks were seen to consist of a stippling of brown. The lighter bordering streaks did not elevate the vessels of the retina, and seemed to be simply a slight opacity of that membrane.

The patient was kept under observation four months and then left the city permanently. Preparations of iron were given and later iodide of potassium, and no fresh hemorrhages occurred. No change was observed in the condition of the fundus except in and near the fresh hemorrhages.

The large hemorrhage became absorbed from below, the blood collecting at its sharp upper margin, the narrow streak of blood extended upward from it, and the new double streak developed just above it, extending in a general direction upward toward the disc, as if there were currents in the retina running centripetally.

Most of the older streaks ran from the periphery toward the centre, as in the cases previously reported, and were gradually lost in the neighborhood of the disc. Some of these older pigmented streaks arose in the periphery from whitish membranous patches, like those found in retinitis proliferans, and one of these patches gave origin to two streaks. Thus these old striæ would seem to have developed like the new one, apparently by the ele-

ments of a peripheric hemorrhage being carried toward the disc in a path lying in the deep layers of the retina.

In studying cases of so-called retinitis striata, one is struck not only by the similarity in course and conformation between the white stripes seen in this affection, and the pigmented angioid streaks, but also by the presence of patches of opacity or of pigmentation of the retina, suggestive of previous hemorrhages, from which many of the white striæ arise.

The cases of retinitis striata that I have myself examined are as follows:

Case 1.—Miss M., aged twenty-five, noticed a blurring of vision three weeks before the examination. Two days previously she found that the sight of the right eye was very poor, and examination of the field of vision revealed a central scotoma. In the inferior portion of the fundus were long stripes, and a network of glistening whitish bands lying beneath and elevating the vessels of the retina. One of these stripes ran through the macula and ended in a line of pigment. Near the macula were small pigmented patches, and to the nasal side of the disc were larger patches of rarefaction and pigmentation. The retinal veins were enlarged.

Case 2.—A boy, aged twenty, following an acute nephritis, had swelling of the discs with white patches in the region of the macula, and hemorrhages scattered through the fundus. Six months later the changes in the disc and about the macula had disappeared, but some of the inferior retinal vessels were bordered with white lines, and in the inferior periphery of the fundus in both eyes were long white stripes ending in patches of pigment.

Case 3.—Miss F., aged thirty-seven, always myopic, noticed a sudden blurring of vision six years previously, which cleared again. Later there had been a gradual failure of sight, and at the examination the patient was only able to perceive the motion of the hand in the lower outer portion of the field. In the upper portion of the fundus were three narrow yellowish-white lines bordered by black, lying beneath the retinal vessels without elevating them, radiating from the neighborhood of the disc and ending peripherically in patches of slight opacity of the retina.

In the lower portion of the fundus was a stippled black stripe running horizontally, and there was increased pigmentation of the surrounding retina. The condition in the other eye was similar, but less extensive.

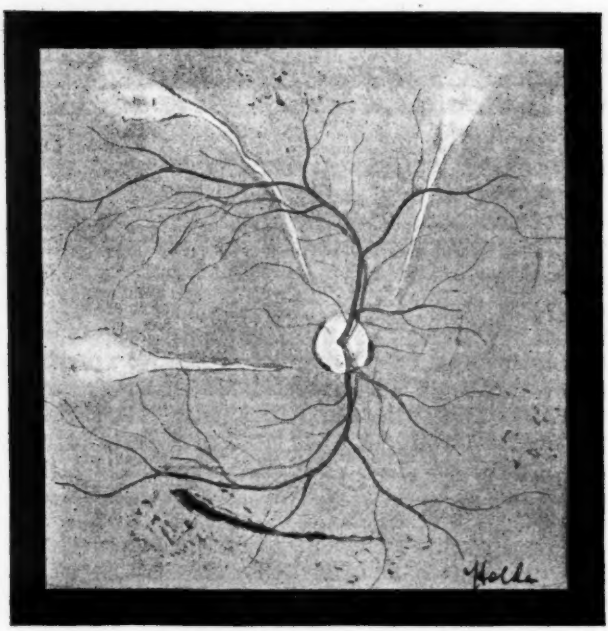


FIG. 2.—RETINITIS STRIATA.
Stripes of rarefaction and one of pigmentation.

In one of the cases pictured in Jaeger's atlas, two of the white stripes originate in dark-brown patches in the periphery, and run toward the disc.

Finally, not to multiply cases further, there is a drawing by Caspar in the Helmholtz Festschrift, in which there is a network of white stripes with black margins beneath the vessels of the retina, and near the disc several long branched hemorrhagic stripes, differing from angioid streaks only in their bright-red color. In looking at these, it would seem

natural that there should have been suggested to the observer the probable hemorrhagic origin of the older white stripes, though Caspar considered the affection choroidal, with possibly some involvement of the retina.

Onesi,[1] writing in 1890, gave abstracts of nine cases of retinitis striata from literature, and in his general description of the affection says: "The light stripes lie beneath the retinal vessels and in front of the pigment epithelium, the choroid not being involved in uncomplicated cases. The pigment may be in the form of masses, or in granules bordering the light stripes. The stripes, often branching, extend from the periphery and gradually fade away near the disc, where the retina has sometimes lost its transparency. The stripes are broader in the periphery, and often end in gray or pigmented patches, or in shallow detachments of the retina. The stripes may or may not elevate the retinal vessels. The macular region may show light spots, white or red stripes, black patches, or rarefaction."

Thus the two affections, angioid streaks and retinitis striata present a remarkable similarity, differing mainly in the color of the stripes, and we know that the ultimate result of a retinal hemorrhage may be a patch of light color or one of pigmentation. In both these affections there are found changes that are known to be due to hemorrhage, while in both, changes that are known to be due to inflammation are for the most part wanting.

It would therefore seem warrantable to assume as a hypothesis, to be verified by future observation, that the affection called retinitis striata, like that called angioid streaks, arises through the elements of peripheric hemorrhages being diffused in a linear manner in the deep layers of the retina and undergoing various sorts of metamorphosis.

While it is probable that the diffusion may take place, in part at least, through previously existing lymph-channels, our knowledge of the lymph currents in the retina is still too indefinite to throw any light on the subject.

[1] *Mittheilungen aus der Oph. Klinik in Tübingen*, 1890.

THE LENS-STAR FIGURE OF MAN AND THE VERTEBRATES.

By PERCY FRIDENBERG, M.D.

(*With 21 figs. on plate.*)

THE structure of the crystalline lens has been the object of so frequent scientific study, that it would seem to offer, at the best, an unpromising field for histological investigation. However, in spite of our accurate knowledge of most structural details of the crystalline body, we are still insufficiently informed as to the composition and proportions of an interesting and possibly important part of the lens, the inter-fibrillary cement or lens-star substance.

Since the early publications of Young, Werneck, Bowman, and Hannover, which were principally devoted to a study of the course of the lens-fibres, little has become known as to the form and composition of the lens-star figure. Nunneley first described the lens-star of vertebrates, and showed that the tri-branch was the type of this order. His view was supported by Huschke, and has been accepted by all later writers. In the human embryo, and at birth, the lens shows no change from this typical form; the three-pointed star of the vertebrates appears in its original simplicity. With the development of the organism and the increased bulk of the lens, however, a change takes place in the arrangement and number of the star-points. In a former publication[1] I reported the results of an investigation on this subject, in the course of which I was able to study the details of the lens-star during life in a large number of normal human eyes.

[1] *Ueber die Sternfigur der Krystall-Linse*, I.-D., Strassburg, 1891.

My observations convinced me that the schematic representations familiar to us from perusal of the anatomical text-books and monographs, and generally accepted as correct, are not reliable, and that these drawings differ quite materially from the form of the lens-star as actually seen *intra vitam*. The theory that the change in form of the lens-star with the development of the individual would become evident as an increase in the number of its rays alone, led to the acceptance as typical of a mathematically subdivided star-figure of six, nine, or twelve rays.

Although this presumption had no basis in fact, and was unsupported by actual observation, these geometrically accurate figures were copied with unquestioning faith by one writer after another, and we find them unchanged in the works of Werneck, Huschke, Fr. Arnold, and others (*vide* Plate p. 159, Figs. 1–5).

The importance of an accurate knowledge of the true form of the lens-star warrants a brief recapitulation of the results of my investigation. The examination was made with the corneal loupe of Zehender-Westien, and the eye so placed that the anterior part of the lens was illuminated obliquely. The rays from the source of light were totally reflected from the anterior surface of the lens, which then presented a bright slate-gray reflex, allowing the details of its structure to be studied with ease. I found, first, a decided variety in the number and arrangement of the star-rays. The regularity of form which I had expected was conspicuously absent. In only one or two cases was I able to find a three-pointed star. Of one hundred lenses about one fifth showed four rays, two fifths presented a five-pointed star, one fifth a figure with six rays, the rest (seven cases) a seven-pointed star. I found no figures with more than seven primary rays, although secondary lines could be counted up to the limit of visibility, making the total of rays twenty or more. The figure at the pole of the lens is rarely that of a true star, as the angles between adjacent rays are usually unequal; besides this, the rays do not issue from the pole or from a point which corresponds to it approximately, but start, usually, from a sutural line running through the pole.

This line is in most cases vertical, but not at all infrequently oblique. Rays proceeding from the opposite ends of such a line produce a figure which appears like an inverted and an upright embryonal tri-branch, in which the vertical rays have joined at the pole.

The division of the rays was quite unlike that usually represented. The difference between the lens-star of the adult and that of the embryo does not consist alone, or even principally, in a dichotomous division and multiplication of primary rays. Dichotomous division is by no means the rule, as a ray may divide into two, three, or more secondary lines, while its course to the periphery is free from divisions on the other side. At times the division of rays is dendritic in character, and resembles somewhat the arrangement of retinal vessels (Laqueur), as the subdivision of rays is most active in the portions of the lens above or below the pole, and the lateral quadrants are less abundantly supplied (Figs. 6–9). An examination of seventy-seven lenses to determine the relative number of rays in the different quadrants of the lens, showed that on an average there were in the upper quadrant two or three, in the lower quadrant three or four, primary rays, with one or two in the nasal quadrant and one ray in the temporal.

Although the quantitative relations of the rays varied somewhat, it was almost invariably found that the number of rays became successively less in advancing from the inferior to the superior quadrant, and then from the nasal to the temporal. The relative frequency of the rays in the upper and lower quadrants varied within certain limits, so that the proportion became, at times, 4:3, 3:2, 5:4, in favor of the former. The relative paucity of rays in the temporal quadrant was constant and quite striking, the total number of rays here being in the proportion of 2:3 in the nasal, of 1:3 in the superior, of almost 1:4 in the inferior quadrant, and as 1:13 in relation to the total number of rays in the three other quadrants.

In regard to the lens-star of mammals I was able to find only sparse observations, and it is the object of this paper to report the result of a study of its structure in the lens of

domestic animals. I have limited myself to lenses of the ox, horse, sheep, pig, dog, cat, mouse, rabbit, deer, and of several varieties of fishes, and in one or two cases have been able to compare the star seen *intra vitam* with the picture presented after staining the extracted lens. The following methods were employed to fix and stain the figures. An aqueous solution of Prussian blue was injected peripherally beneath the lens-capsule, and the excess of stain washed away in distilled water, leaving the star-figure outlined in deep blue.

This procedure was employed in the case of larger animals. The smaller lenses seemed too delicate to allow them to be subjected with impunity to this mechanical method. In this series I injected a solution of ferrocyanide of potassium into the anterior chamber of the living animal, and after death subjected the lens in its capsule to the action of ferric chloride in aqueous solution. The results of this method were not uniformly satisfactory, as the resulting blue stain sometimes became so intense as to obscure the details of the lens surface.

The most satisfactory reagent was found to be nitrate of silver in the strength of 1:500–1:1000. Robinski,[1] who claims that the lens-star is invariably an artificial product due to the irregular shrinking and cleavage of the lens surface after hardening in Müller's fluid, alcohol, and similar reagents, considers the silver solution free from all objection on this score, as it has no hardening or macerating action on the albumen of tissues. On this point he says: "I must call attention to the fact that imbibition takes place when the lens is immersed in hardening fluids. As the superficial layers of this body contain a greater percentage of water than the deeper (Kölliker) there is more absorption of fluid in the dry nuclear layers than in the watery cortex; the nucleus itself imbibes most freely, and exerts a bursting strain on the layers enveloping it. This is followed by a fissuring of the surface of the lens, with the production of so-called star-figures."

In his investigations, Robinski used nitrate of silver in so-

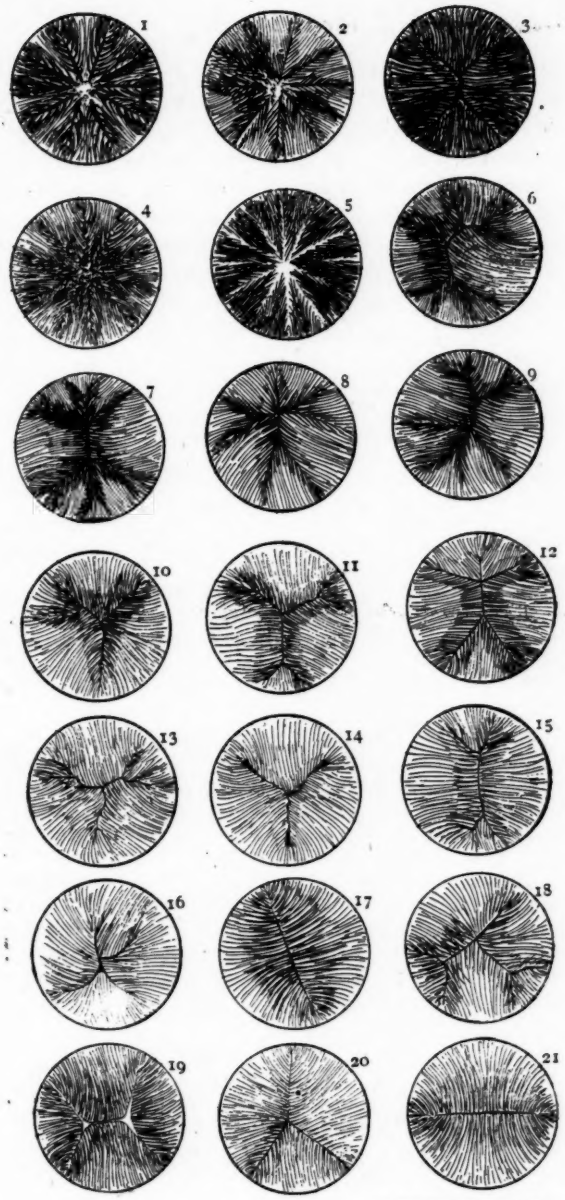
[1] *Med. Centralblatt*, 1888.

lution (1:800), the fresh lens being immersed in this fluid for from five to fifteen minutes. He claims that no trace of a lens-star is seen in lenses treated in this way, which is the only process free from the objections raised against hardening fluids, as it leaves the surface of the lens unchanged. The silver in weak solution has no action on the lens fibres, but makes their contour visible, as a precipitation of metallic silver takes place along the lines of contact in the form of minute brownish-black granules. At first I intended to use this method for the study of the lens fibres, as it had been expressly stated by the above mentioned observer that he had found no trace of a lens-star. I immersed fresh lenses, in the capsule, in a weak solution of silver nitrate, and within five or ten minutes could see clearly the outlines of the lens-star figure, which gradually grew more and more distinct as the precipitation of silver progressed. In each case a drawing of the figure was made, and the lens left to the action of the silver for twenty-four hours.

Meanwhile the entire surface of the lens took on a rich chocolate color, the rays of the star-figure having become intensely black and offering a strong contrast.

There were absolutely no fissures or even the slightest break to be discovered on the smooth, highly reflecting surface of the lens. Microscopic examination with low powers also showed the surface to be perfectly intact, so that we may well assume that there has been no imbibition or manifestation of a "bursting strain," and that there is no doubt of the star-figure being a natural structure which can be made visible by the simplest methods. The results of this staining method are equally satisfactory for small or large lenses. The lens-star of mammalians corresponds in general to that of man. In the young the tri-brachial type of the vertebrates is seen unmodified (Fig. 10), while in fully developed animals the rays are more numerous (Figs. 12, 15, 18), somewhat irregular in distribution, and may arise, as in the human lens, from a sutural line instead of meeting in a common pole (Figs. 12, 15, 19).

The calf lens (Fig. 10) shows small lateral twigs running from the primary rays of the figure, which resembles the condition seen in man. Ox lenses (Figs. 11, 12) show a



P. FRIDENBERG, del.

more complicated star than that of the calf, although the three-point figure is still evident. The irregularity in distribution of primary rays, with the increase in number of the branches, and the relative prominence of lateral twigs are particularly noticeable. The lens of the mouse (Fig. 16) shows a three- or four-rayed star with curved branches presenting their convexity to the pole. The lens of the rabbit (Fig. 17) differs from the others examined in presenting no star proper. This figure is replaced by a sutural line running through the pole, from above usually to the lower outer quadrant. A similar line takes the place of a star-figure in the lens of fishes (Fig. 21).

EXPLANATION OF THE DRAWINGS.

Figs. 1–5. Schematic representation of a theoretically constructed lens-star.

Fig. 1. After Huschke, Edw. (Meckel's *Archiv*, 1832, Fig. V.).

Fig. 2. After Werneck (Ammon's *Zeitschrift*, vol. iv. and v., Fig. V.).

Fig. 3. After Arnold, Fr. (*Tabulæ Anatom.*, Fascic. ii., 1851, Fig. 17).

Fig. 4. After Arnold, Fr. (*Tabulæ Anatom.*, Fascic. ii., 1851, Fig. 18).

Fig. 5. After Arnold, Fr. (*Tabulæ Anatom.*, Fascic. ii., 1851, Fig. 19).

Figs. 6–9. Star-figures of the human lens as seen with the corneal loupe, showing the origin of secondary rays from a sutural line and their distribution in the different quadrants.

Fig. 6. Left lens. Adult (thirty-three years).

Fig. 7. Left lens (sixteen years). Frequent form.

Fig. 8. Left lens (thirty-two years). " "

Fig. 9. Right lens (twenty-eight years).

Figs 10–21. Star-figures of the lens of animals treated with nitrate of silver, 1:500.

Fig. 10. Lens of calf.	Magnified	1.5x.
Fig. 11. Lens of ox.	"	1.25x.
Fig. 12. Lens of ox.	"	1.25x.
Fig. 13. Lens of sheep.	"	1.75x.
Fig. 14. Lens of pig.	"	2x.
Fig. 15. Lens of cat.	"	2x.
Fig. 16. Lens of mouse.	"	10x.
Fig. 17. Lens of rabbit.	"	2.5x.
Fig. 18. Lens of horse.	"	1.25x.
Fig. 19. Lens of buffalo.	"	1.25x.
Fig. 20. Lens of deer.	"	1.5x.
Fig. 21. Lens of salmon.	"	3x.

A TUMOR OF THE CILIARY BODY; WITH REMARKS.

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Translated by Dr. WARD A. HOLDEN.

(*With Plate VI., Vol. XXVIII., Germ. Ed.*)

M. W., a man of sixty-six was first seen early in September, 1893, when he complained of having had a dull feeling of pressure in the right eye for four months. Vision was reduced and he saw halos. There was + T ½; and in the lower outer quadrant of the chamber angle, a gray stripe of foreign matter, 0.5 *mm* wide, separated the periphery of the iris from the ciliary body and projected forward above the level of the iris. The upper inner half of the posterior capsule of the lens was diffusely opaque. Behind the lens and corresponding in position to the foreign substance at the periphery of the iris was a rounded blackish tumor the size of a pea. Fundus normal; V = ⅓.

Sept. 23d.—A brown point was seen under the conjunctiva 2 *mm* from the limbus, in the middle of the lower outer quadrant of the sclera, corresponding in position to the point of perforation of an anterior ciliary artery. The mass at the periphery of the iris was broader, and the pupil had assumed a kidney form, due to muscular paralysis in the affected portion of the iris.

Dec. 28th.—There was a more marked iridodialysis caused by the tumor, and the pupil was displaced upward and inward. The projecting tumor mass, 2 *mm* broad by 6 *mm* long, was of white color. The lens was more opaque; the tumor mass touching it was larger, more rounded, and of dark-gray color. The sclera nodule was larger.

The patient had an attack of iritis., relieved by atropine. The lens became more cloudy, the vision was further reduced, and the scleral nodule increased in size. The ball was enucleated and hardened in Müller's fluid.

Macroscopically there is a loaf-shaped tumor in the lower anterior ciliary region, 9.5 *mm* long and 4.5 *mm* broad—Fig. 1. Posteriorly it passes gradually over into the normal choroid, anteriorly it is broader and pushes the iris insertion 3 *mm* inward and projects into the anterior chamber. The tumor is slightly mottled with pigment. The lens is displaced, its margin adjacent to the tumor being flattened, and the capsule being adherent to the tumor.

Macroscopically the tumor is seen to be composed of long and short spindle-cells, running for the most part meridianally, but some bundles having an equatorial course, and a few running in the direction of the radiating portion of the ciliary muscle. The nuclei are mostly spindle-shaped, but some are rod-shaped or oval—Fig. 2. In transverse section the nuclei are mostly angular, and not round. The cells form compact bundles of different length and thickness, which divide in various ways and form a meshwork. The groups of bundles are separated by connective-tissue septa, but the septa are rarely found between individual bundles. Toward the choroid there is a more marked pigmented reticulum giving the tumor an alveolar appearance. In the periphery of the tumor the cells as well as the nuclei have an oval form. The older cells in the substance of the tumor are for the most part spindle-shaped, and many of these long spindle-cells resemble, in longitudinal section, unstriped muscle-cells. These cells lie mostly isolated in the neighborhood of blood-vessels. Some cell-bundles that are cut transversely also show a similarity to leiomyoma in the multiangular contour of the cells and in their grouping into bundles—Fig. 2.

Posteriorly the tumor is covered by the pigment epithelium and the pars ciliaris retinæ, but anteriorly it breaks through these. The tumor contains many blood-vessels. The ciliary processes are not recognizable except in a small portion anteriorly. The lens capsule near the equator has

been detached and is adherent to the tumor—Fig. 1. The epithelium here is replaced by a layer of fibrous tissue several cells deep.

The iris appears as a continuation of the anterior portion of the tumor, the latter being intercalated between the iris and choroid. The tumor has invaded the tissue of the iris, however, and both the pigmented and unpigmented cells have proliferated and apparently form sarcoma cells.

The episcleral nodule consists of long spindle-shaped cells with rod-shaped nuclei, in thin layers between the scleral lamellæ, strongly resembling ciliary muscle. Pigmented cells could be followed from the primary tumor to the episcleral nodule along the anterior ciliary vessels.

In its clinical and macroscopic aspects this tumor does not differ from the thirty cases in the literature (Mules, *Trans. Ophth. Soc. Un. K.*, 1888), all being loaf-shaped or hemispherical, springing from a particular portion of the ciliary body, and soon leading to iridodialysis, opacity and dislocation of the lens, and later to the formation of episcleral nodules. They are mostly composed of long spindle-cells in interlacing bundles, and are irregularly mottled with pigment.

The similarity of all these primary tumors of the ciliary body makes it seem probable that they all belong to a single tumor-group.

In only six cases were variations of this tumor-structure found, the spindle-cells, all or in part, being considered unstriped muscle-cells, and the terms myoma and myosarcoma of the ciliary body being used to designate the growths.

The more one works in pathological histology, the more one becomes convinced of the difficulty in a given case of distinguishing the pathological formation of unstriped muscle-cells from spindle sarcoma-cells and from young fibre-cells of fibrous tissue. Virchow (*Die Krankhaften Geschwülste*, vol. ii., p. 199) says that it is particularly difficult to distinguish spindle sarcoma-cells from unstriped muscle-fibres, and to separate certain varieties of myoma from spindle-celled sarcoma. It is easy to recognize the unstriped muscle-cells in the vessel-walls, uterus, and ciliary body in normal

conditions, but it is almost impossible to distinguish isolated normal muscle-cells from isolated spindle sarcoma-cells, and the difficulty is increased in pathological cases.

In myoma one might think that the manner of arrangement of the cell-bundles and the numerous delicate connective-tissue septa that are so well seen in normal and hypertrophied uterus tissue and also in the ciliary body might be of assistance in making a differential diagnosis, but these conditions are not constant. The tissue from which the tumor arises and the wavy or even serpentine form of the cell-nuclei are points of some value in diagnosis.

After these general remarks based upon the study of unstriped muscle in normal and pathological conditions, of myomata and fibromyomata of the uterus, and of various forms of spindle-celled sarcoma, we shall examine the six cases of spindle-celled tumor of the ciliary body reported as myomata and myosarcomata, in order to see whether unstriped muscle-cells were really present.

The first case is that of Iwanoff-De Wecker, in the year 1867, which was first described as a pure myoma and later as a myosarcoma, one regarding it as exclusively muscular, the other as principally muscular. "The entire outer portion of the tumor toward the sclera is composed of spindle-cells with rod-shaped nuclei. In the middle portion the muscle-fibres run longitudinally. Among the muscle-fibres are numerous connective-tissue cells. Toward the vitreous, spindle-cells and branched connective-tissue cells predominate, making up nearly the entire inner portion of the tumor." No drawings were given.

In Salomon's case (*Trans. Ophth. Soc. Un. K.*, vol. ii., p. 263, 1882) Priestley Smith examined the tumor and stated that it had the microscopic character of a myoma or myofibroma, consisting of spindle-cells mostly arranged in the form of reticular fibrillæ and running parallel, presenting a great similarity to the tissue of the normal ciliary muscle. A committee, composed of Brailey, Mackenzie, and Nettleship, made the following report on the case: "The tumor consists of fine cells with rod-shaped nuclei, which cannot be distinguished from the cells of the ciliary muscle or the cells

of a 'fibroma' (myoma) of the uterus, with sections of which it was compared. The arrangement of the cell-fibres in long bundles, which take principally an antero-posterior direction, but also run obliquely, is entirely similar to the relations of the normal ciliary muscle." No microscopic drawing is given.

The argument that a spindle-celled tissue with parallel fibres resembles the normal ciliary muscle, and that its spindle-cells resemble those of a fibroma and a myoma of the uterus, is not sufficient to convince me that the tissue was myomatous. For so many varieties of spindle-celled sarcoma tissue may resemble ciliary muscle, and it is the less convincing when the same tissue resembles uterine fibroma, fibromyoma, and myoma, tumors composed of connective-tissue-fibre cells that differ considerably from one another. And incidentally it may be remarked that Billroth even in 1880 wrote (*Die allgem. chirurg. Pathol. u. Therapie*, Billroth-Winiwarter, ix. edit., p. 718) of the fibro-myomata of the uterus, that it would be more natural to class these tumors with the sarcomata.

Mules's case (*l. c.*, p. 66) of myosarcoma of the ciliary body, reported in 1888, was examined by Brailey, who said: "The neoplasm consists of elongated spindle-cells with slender nuclei, the cells forming a network of bundles with occasional large spaces between them; toward the surface of the tumor, particularly the inner, the cellular tissue is denser and the nuclei more numerous, and the reticular arrangement can hardly be made out, and the cells here can scarcely be considered spindle-cells. The reticular appearance is similar to that of a cavernous tumor, but I believe that the tumor is allied to unstriped muscular tissue, and that it has taken its origin from the muscular tissue of the ciliary body, the structure of which it resembles." Dreschfeld and Harris regarded the tumor as sarcomatous, and Robinson thought it a myosarcoma. No drawing was given.

The only spindle-celled tumor described as a pure myoma was that of Lagrange in 1889 (*Arch. d'opht.*, ix., p. 484). In meridional sections almost all the muscle-elements are cut transversely. Between the bundles which these fibres

form are groups of dark-stained embryonic cells. In sections demonstrated by Ferré, quantities of young embryonic cells in rapid growth, identical with sarcoma cells, were found. This, however, seemed to be accidental, and Lagrange found them in none of his sections. Besides small islands of connective-tissue cells the sections contain only muscle-fibres and blood-vessels. The muscle-fibres form bundles separated by broad spaces. Lagrange believed that the meridional fibres of the ciliary muscle were preserved, and the radial portion of the fibres also did not take part in the pathological process. All the neoplastic fibres are situated internal to the posterior portion of the meridional fibres and in the anterior portion of the choroid. They represent an abnormally large radiating muscle which from Müller's muscle is distributed through the entire thickness of the ciliary muscle. Two drawings are said by Lagrange to represent unsatisfactorily a pure myoma, with which I quite agree.

Deutschmann says of his case of myosarcoma of the ciliary body in 1890 (*Beiträge zur Augenheilk.*, i., p. 72) that the tumor consisted of tracts of cells regularly arranged in a longitudinal direction, many of the spindle-cells being undoubtedly sarcoma cells, while many other tracts of cells were identical with unstriped muscle. No microscopic drawing is given.

Lange (*Graefe's Archiv*, xxxvi., p. 247) describes his case of myosarcoma of the ciliary muscle in 1890 as follows: "The tumor consists of delicate spindle-cells with rod-shaped nuclei. Most of these cells are arranged in meridional bundles. In the central portion are many bundles cut transversely, the cells of which then appear round or angular with small round nuclei. These parts appear alveolar. The nuclei are slightly clubbed at the ends, and the cells are distinctly larger than the unstriped muscle-cells of the normal part of the ciliary muscle." Lange says further: "On account of the great difficulty in distinguishing unstriped muscle-fibres from other spindle-formed connective-tissue cells it was not easy to make the diagnosis here. After careful study I came to the conclusion that my case was one con-

sisting of hypertrophic and hyperplastic unstriped muscle-fibres and spindle-formed connective-tissue cells, originating in the anterior part of the ciliary body, and the diagnosis myosarcoma seems justifiable." An instructive drawing is given of the longitudinally and transversely cut bundles of spindle-cells with rod-shaped nuclei composing the tumor, but it does not convince us that muscle-fibres are a constituent of the tumor.

There is a natural tendency for every one who finds a tumor of the ciliary body with spindle-cells and rod-shaped nuclei to believe that he has to do with a myomatous proliferation of the ciliary muscle. This was true in my case, and my view of the nature of the tumor oscillated between myomatous, sarcomatous, and myosarcomatous, until I had made a more careful study of spindle-celled sarcoma in general and uterine tumors in particular, and had found in the former complete analogues of the tumor structure in my case, which I must, therefore, consider a fascicular spindle-celled sarcoma.

My experience, together with the mostly insufficient histological description of the other cases, with the lack of drawings, and the fact that often different observers differ in their opinion of the same tumor, has led me to the conclusion that the designation of the six tumors as partially or altogether myomatous has arisen more from the subjective conception of the authors, and is not objectively correct.

According to my conviction, the reported cases of spindle-celled tumors of the ciliary body which conform to a definite clinical and anatomical type are all to be considered spindle-celled sarcomata; and the fact that they have been in some cases considered myomata or myosarcomata is due chiefly to the great similarity of the individual spindle-cells and their grouping, to unstriped muscular tissue, and to the fact that they have their origin in a tissue rich in unstriped muscle.

EXPLANATION OF THE FIGURES.

Fig. 1.—A quadrant of the ball with the mottled tumor, the episcleral nodule, the adhesion of the lens-capsule, the partial

absorption of the cortex of the lens, and the break in the pars-ciliaris retinæ. Magnified six times.

Fig. 2.—A portion of the middle of the tumor, with alternating longitudinal and transverse sections of the spindle-cells, the latter grouped into bundles by secondary sheaths.

Fig. 3.—The point of adhesion between the detached and wrinkled lens-capsule and the fibrous tissue and swollen cells which are products of the altered lenticular epithelium.

THE ANTERIOR OUTFLOW CHANNELS OF THE EYE AND THE ARTIFICIAL PRODUCTION OF GLAUCOMA.

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(*With Plate IX. of Vol. XXVIII., Germ. Ed.*)

THE controversy with regard to the passages by which the fluid of the tissues makes its exit from the interior of the eye has continued up to the present time. The only statement that is generally accepted is that the aqueous has its outlet "in front," and that a portion of the liquid of the vitreous gets out of the eye through the lymph-channel of the optic nerve.

In coming to a decision with respect to the experiments bearing upon this point, we must differentiate two things actually distinct, viz.: (1) the *path taken by the body of liquid as a whole*, this passing according to natural laws from the regions of higher pressure (the capillaries) through the tissues and their interspaces into the preformed passages of outflow (the lymph-vessels); and (2) the *channels along which dissolved substances* (including gases) *undergo diffusion.* Such dissolved substances would spread to all parts from which they were absent before or in which they were present before in a less concentrated state.

In experiments upon the path taken by the body of liquid as a whole, we must, for reasons that can be readily appreciated, use substances which are not normally, nor, in fact,

under any conditions, present in the eye. In a perfectly dead eye such substances would diffuse uniformly in all directions according to purely physical laws, being affected only by the different degrees of facility for imbibition and diffusion afforded by the tissues. In the living eye this state of things is modified by the natural "tissue-current," and the same is true, though to a less degree, for the recently excised eye, provided that the tissues have not become entirely dead, and all differences in pressure have not been equalized.

Many years ago experiments that I made (Virchow's *Archiv*, vol. lxv., p. 401, and *Arch. f. Augen. und Ohr.*, vol. vii.) proved that potassium ferrocyanide introduced into the cavity of the vitreous passed very readily into the anterior chamber and then left the eye by the same channels as do substances that are introduced directly into the anterior chamber. For the study of these latter channels of exit, in fact, injections into the vitreous cavity are more suitable than injections into the anterior chamber, since in the former case the walls of the latter remain perfectly intact. Hence when I undertook to make a series of experiments intended to demonstrate the effect that substances which, though aseptic, are yet provocative of inflammation, exert upon the anterior channels of outflow in the eye, I determined to introduce these substances into the vitreous. Since in this case the substances employed were such as do not normally circulate in the currents by which the tissue-metamorphosis of the eye is effected, precisely the same conditions were present here as with the injections of potassium ferrocyanide. The latter, in fact, give us the place where we are to look for the effects that substances provocative of inflammation will excite, when introduced into the vitreous.

The experiments were made upon dogs because their eyes, as far as the parts here under consideration are concerned, correspond more closely to human eyes than do those of rabbits, which are generally used. Before describing these experiments, however, I consider it absolutely necessary to describe the channels by which the ferrocyanide introduced into the dog's vitreous leaves the eye. To effect this from one to three drops of a solution of potassium ferrocyanide

were introduced as nearly as might be in the centre of the vitreous and, after a varying interval of time, the animals were killed. The eyes were at once enucleated, opened, cut in two, and exposed to the action of ferric chloride dissolved in dilute alcohol. By the blue coloration which is immediately developed, the mode of distribution may then be recognized.

It is evident, in the first place, that this solution undergoes practically no diffusion backwards, but does diffuse into the anterior portions of the vitreous. Spreading from this locality it colors the contents of Petit's canal and of the anterior chamber. It is only later, after six or eight hours' action, that some of the coloration penetrates the lens, beginning at the posterior pole and extending into the posterior cortex.

Generally the ciliary body is also found to be more or less strongly tinged with blue, and so is the adjoining peripheral portion of the iris—the portion constituting Ulrich's peripheral channel of transmission through the iris—which I (*l. c.*) discovered and described much earlier than he did, although I gave a different interpretation to it. In this case the ferrocyanide is found also in the anterior chamber and in such abundance that it seems to me far more probable that the coloring matter had penetrated from Fontana's space than through the iris.

The first stage in the passage of the coloring matter through the wall of the eyeball is manifest after two or three hours under the form of the well-known and much-discussed blue streak in the corneo-scleral junction (Fig. 1). This streak as it makes its way backwards lies farther and farther out in the sclera, and ultimately, at some point behind the equator of the eye, empties into the lymph-space of Tenon's capsule.

This condition of things, which is represented in Fig. 1, is the easiest to obtain, and most observers who have repeated my experiments, seem to have got this stage and nothing more. Much more instructive, however, are the conditions obtained after a somewhat longer time, the most characteristic of which are represented in Figs. 2, 3, and 4.

For, while the streak in the corneo-scleral junction and in the sclera remains as it was, the hindmost layers of the cornea also become colored (Fig. 2), and later on the entire cornea takes on a blue tinge, and the color spreads not only backwards into the sclera, but also into the subconjunctival connective-tissue, in which the main lymphatic vessels run in the direction of the four recti muscles to the angles of the eye and to the retrotarsal folds. We then get pictures like those which Fig. 3 represents.

Later still we find that only the anterior layers of the cornea are colored, the coloration extending from them into the subconjunctival tissue (Fig. 4). The blue streak in the sclera, though still present, does not now extend as far forward as the anterior chamber.

After from eight to ten hours no more ferrocyanide can be found in the eye.

Leber and others, including recently Gifford (these ARCHIVES, vol. xxi., No. 2) regard the results obtained by my method as of little value because of the precipitates produced at the spots where the two reagents first come into contact. But owing to the way in which the eye is cut into and bisected, the reaction is made almost instantaneous. Moreover, I do not know of any other way of fixing the spot to which a substance contained in the eye has penetrated.

From an unprejudiced consideration of Fig. 3, and especially of Fig. 4, there can be no doubt that during life the ferrocyanide had entered the cornea from the anterior chamber. And, as I showed in my earliest papers, the endothelium of Descemet's membrane is not an absolute bar to the passage of dissolved substances from the anterior chamber into the cornea, but is only a means of regulating the passage, since it prevents more from entering than can under normal conditions flow off again at the corneo-scleral junction.

The experiments whose results have been thus shortly summarized were made and published as much as fifteen years ago.

Following are the positive facts resulting from my experi-

ments with respect to the conditions of outflow in the anterior segment of the eye:

1. The aqueous is secreted not only by the ciliary processes, but, to a slighter extent, also is derived from the posterior segment of the eye. Substances which are not normally present in the latter, *i. e.*, pathological substances, are especially apt to get into the aqueous.

2. The outflow from the anterior chamber takes place not only (although it does mainly) from Fontana's space, but a portion of the aqueous, even when the endothelium of Descemet's membrane is intact and the intra-ocular pressure is not heightened, penetrates into the cornea itself and flows in the direction of the subconjunctival tissue. This is particularly the case with substances which are not normally present in the aqueous (and in the anterior portion of the vitreous).

3. Dissolved substances present in the anterior portions of the vitreous also get into the lens, and first into its posterior cortex.

In all these cases it is not a question of preformed closed channels of exit but of a process which goes on in connective-tissue spaces and in the matrix of the tissues. The fluid of the eyes does not get into closed spaces which are lined with endothelium until it reaches the cavity of Tenon's capsule and the efferent lymph-vessels in the subconjunctival tissue.

What has been said holds good only for *dissolved* substances. To solid and formed matters the lens-capsule and Descemet's membrane are perfectly impervious; such matters must either pass through these membranes in a state of solution, or a breach of continuity in the membranes must have taken place. The same is true for cellular elements.

In experiments that I have made in recent years by introducing *into the vitreous aseptic substances provocative of inflammation*, I did not so much concern myself with the changes that these substances excite sooner or later in the posterior segment of the eye; these have been sufficiently described by Leber. My main purpose was to investigate their action in the *anterior* segment of the eye.

Since the matters injected were not traceable directly, and since the changes that resulted from their action required time to make them visible, the latter could be expected to take place only after the lapse of some considerable time. Moreover, I did not intend to produce a transient action which could hardly be demonstrable anatomically, but wished to introduce a difficultly soluble agent which would exert an effect for a longer time. I thought to imitate the conditions exhibited by a malignant growth developing in the vitreous and secreting irritating substances which give rise to reactive inflammation of the tissues; and *hoped at the same time to produce an artificial glaucoma.* I succeeded only to a certain extent, because the reagents used did not secure the permanent effect that I had hoped for and because they acted rather differently than I had expected. Nevertheless, the results are interesting enough, although their main value lies in their clinical and not in their pathologico-anatomical aspect.

The experiments were all made upon dogs under morphine narcosis; and it may be remarked in passing that in this animal the pupil is ordinarily not contracted under these conditions. The eyes were hardened in Müller's fluid and alcohol and imbedded in paraffin, and the sections were stained with hæmatoxylin.

In experiments I. and II., oil of turpentine was injected into the vitreous. This produced detachment of the retina and posterior polar cataract with accumulation of liquid beneath the posterior capsule of the lens; the formation in the anterior chamber of gelatinous exudate which was soon absorbed; and the development of an amorphous exudate in the vitreous. The second case was complicated by an iritis due to injury of the eye. Apart from the consequences due to this accident the anterior part of the eyeball seemed to be but slightly affected by the injections, and the latter *per se* occasioned no glaucomatous symptoms.

The failure here being ascribed to the undue violence of the reaction, a third experiment was made with an injection of a mixture of 1 part of oil of turpentine and 19 parts of olive oil. In this case the injection produced localized

opacity and liquefaction of the vitreous with subsequent shrinking of the latter and detachment of it from the retina; and, in the lens, rounded opacities in the cortex and distension of the posterior pole by a coagulated liquid containing globular bodies. *Four slight and transient attacks of glaucoma*, two developing spontaneously and two as the result of the instillation of atropine, and exhibiting the characters of the so-called prodromal stage of the acute inflammatory variety of the disease, occurred in the first two months. No permanent changes were produced, however, and in the third month even the instillation of atropine failed to produce an increase of tension; and after four months, when the animal was killed, the structures in the anterior part of the eye were found to be perfectly normal.

In experiments IV. to VII. a mixture of oil of turpentine, 1 part, and olive oil, 2 parts, was used.

In experiment IV. a glaucomatous attack with T + 2 and the formation of a gelatinous exudate in the anterior chamber set in seven hours after the injection. The anatomical conditions found after killing the animal on the following day were marked œdema of the cornea and distension of Fontana's space and of the adjoining parts of the iris and ciliary body with coagulated fibrinous exudate and red blood corpuscles.

In experiment V. the animal was allowed to live for 50 hours. There were attacks of glaucoma, one 10, the other 34 hours after the injection. The latter produced detachment of the hyaloid membrane, and a grayish non-cellular membrane was formed in the vitreous about the injected liquid. Fontana's space showed no pathological alteration, and, except for marked œdema of the cornea, the anterior portion of the eye was normal.

In experiment VI. one pretty severe attack of glaucoma set in 8 hours after the injection. This lasted 2 days, and was not repeated even when atropine was instilled. The animal was killed at the end of 10 days. The main lesions found were cellular infiltration in the network of the ligamentum pectinatum and apparently also of the optic disc, and beginning detachment and shrinking of the vitreous.

In experiment VII. a transitory attack of glaucoma set in 8 hours after the injection. This was followed by a diminution of tension (T – 1). When the animal was killed 11 days afterwards, the cornea was found to be œdematous and had isolated deposits on its posterior surface; there was an infiltration of round cells in the ciliary processes and in, or rather back of, Fontana's space, and there was hemorrhage in the suprachoroid of the ciliary body; and there were thickening and infiltration of the conjunctiva and some infiltration of the retina and optic disc.

In experiment VIII. pure olive oil was injected. A glaucomatous attack followed 18 hours later and lasted till the following day. An exudate also formed in the anterior chamber; but this shrank and was completely absorbed within three days after the injection was made. These phenomena may both be fairly attributed to the effect of the injected material as such; but the subsequent symptoms, which were those of a violent panophthalmitis, must be regarded as the result of septic infection, which was evidently due to the fact that the oil could not be properly disinfected by heating before it was injected.

The results of the experiments just detailed had been unsatisfactory, because in none was a *permanent* aseptic irritation set up in the anterior portion of the eye, such as would produce a continuous glaucomatous condition instead of producing, as in these cases, merely transient glaucomatous attacks, which were of such a character as to simulate those occurring in the prodromal stage of the disease. With no hope of securing a more permanent and slower action, injections were made with a mixture of mercurial ointment (unguntum hydrargyri cinereum 3.0) with olive oil (7.0) and oil of turpentine (6 drops); the idea being that the finely divided mercury in this "oleum cinereum" would pass slowly into the circulatory channels of the eye and produce a continuing condition of obstruction and inflammation. This "oleum cinereum" was applied in cases IX. to XIII.

In experiment IX., in order to follow the action of the material to its end, the case was watched for 11 months.

On the third day after the injection there was a violent attack of glaucoma which lasted 2 days. Simultaneously there was formed in the anterior chamber a gelatinous exudate, which by the sixth day had contracted to one half its original size. On this latter date there were the first signs of an irido-choroiditis which, at first associated with diminution of tension, subsequently led to increase of tension, the formation of an annular posterior synechia and retro-iridic exudate, and in 2 months to phthisis bulbi. From this time on the eye remained perfectly free from injection and irritation; and it is to be noted that during the whole course of the disease, the irritative symptoms remained very moderate, and that no tendency to the development of a sympathetic inflammation in the other eye manifested itself.

In experiment X. the attempt was made to find what were the consequences of the injection in its earliest stages; hence the eye was enucleated 24 hours after the injection had been made. The tissues were mainly normal except for the presence of a coagulum in the anterior chamber[1] and of scattered round cells in the peripheral layers of the vitreous, in Fontana's space, and in the ciliary processes.

In experiment XI. the animal was killed 48 hours after the injection. The aqueous had already become turbid, although the opacities had not yet had time to gather into a coherent mass. The eye, after hardening, showed coagulation of the aqueous and of the vitreous. Round cells were present in the extreme periphery of the latter (in the portion adjoining the retina), and also, and much more abundantly, in the portions next to the ciliary body, and were also found in considerable number in the ciliary body itself, in Petit's canal, and in the vicinity of Schlemm's canal. There was likewise some cellular infiltration of the retina, and the retina at one spot was detached from the choroid by a non-cellular exudate. The anterior chamber contained a cellular exudate only in its peripheral portions.

[1] Produced by the material used for hardening the specimen; but its presence indicative, according to Knies, of a change in the constitution of the aqueous.—TRANSLATOR.

The cornea was normal except for the infiltration about Schlemm's canal.

In experiment XII., also, the animal was killed after 48 hours. A lenticular exudate had already formed in the anterior chamber. The vitreous here too had been coagulated by the hardening agent. The contents of the anterior chamber were likewise coagulated, and except in the portion adjoining Descemet's membrane were almost entirely free from cells. The cornea was normal except for a cellular infiltration beginning at its margin and extending from there into the innermost layers of the cornea and into the subconjunctival tissue. Cellular infiltration was also found in Tenon's capsule, Fontana's space, the ciliary body (which was hyperæmic), and the ciliary processes, and from the latter was continued into the vitreous and into Petit's canal. The amount of infiltration diminished from before, backwards. The retina was not detached, but near the papilla was folded, and in this region an amorphous exudate lay between it and the choroid.

In experiment XIII. the animal was killed after 72 hours. A pair of lenticular exudates had formed in the anterior chamber. There were no glaucomatous symptoms. The condition on autopsy was essentially that found in the preceding case. The chief additional feature was an abundant cellular infiltration, which was seated in the connective tissue surrounding the central vessels of the optic nerve and which extended into the vitreous.

Experiment XIV., which consisted simply in the examination of a normal dog's eye, was made to determine how far the appearances in the foregoing experiments were applicable to the eye of man. The only essential difference between the two, which could have any bearing upon the results already detailed, was that, upon the optic papilla of the dog and surrounding the central vessels, there is a flattened cone of connective tissue, containing numerous cells and attached by its apex to the hyaloid membrane of the vitreous. This finding makes it doubtful whether the cellular infiltration found in some of the cases in the optic disc was altogether abnormal or not. In other respects the anatomical

conditions are the same in the canine and the human eye, and the two therefore, when placed under the same experimental conditions, should react alike and exhibit the same appearances.

It was my object in making these experiments to demonstrate the passages of outflow from the vitreous and aqueous by means of the changes, which substances capable of provoking irritation and inflammation excite in their passage through the walls of the eyeball. The action must vary according as it is vascular or non-vascular tissues that are affected. Direct tissue-changes might perhaps be expected to take place; furthermore coagulation-processes in the channels of outflow and consequent œdema of the tissues back of them; and—most important and most obvious change of all—local inflammation manifested by cellular infiltration of the parts affected.

From the results of the injection-experiments with the potassium ferrocyanide changes were to be expected in the vitreous itself, especially in its anterior segment, in Petit's canal, in the anterior chamber, in the cornea, in the angle of the iris and its vicinity, and later also in the lens. From clinical experience it is likely that the optic papilla also and its vicinity would be involved, it being understood that our investigation deals with only those changes that are the direct consequences of the injections, and not with those that occur weeks and months afterwards. It should also be stated that obvious pathological changes due to the injection are not to be looked for as soon as those that prove the mere presence of an injected material like potassium ferrocyanide; the former requiring a certain time for their development. The results of my experiments are in complete accordance with this fact.

The mildest of the reagents was olive oil, disinfected with oil of turpentine (1:19). After the primary effect, due to its introduction, had ceased, the eye became perfectly quiet again. The more violent action exerted by the unmixed olive oil is explained by the fact that it was not aseptic, and that the germs and their decomposition products which it contained were not destroyed by admixture with turpentine, as in

the other cases. Furthermore, the oil itself, unless perfectly fresh, probably exerts an irritant action, even when it is aseptic, as the products of its *chemical* decomposition (glycerin and fatty acids) are more or less soluble, and decidedly irritant. However, as far as the results of the experiments are concerned, it is a matter of indifference what substance is used for the injections. When a very active material is used, its remote effects come to an end sooner, because the material itself is speedily encapsulated by a membrane produced by coagulation of the tissues in its vicinity.

The first result, clinically speaking, of the injection of dilute oil of turpentine, was, in every case in which the observation could be made at the proper time, an *attack of glaucoma*, setting in after a period of about six hours (rarely more), during which the eye had remained apparently normal. This attack had the character of those occurring in the so-called prodromal stage of glaucoma, and, being always quite transitory in character, escaped observation in cases I. to III., because it had come on and had disappeared during the night. The attack was characterized by the usual signs: marked increase of tension, mydriasis (in spite of the simultaneous use of morphine), and sometimes slight vapor-like opacity of the centre of the cornea.

The fact that the attack did not develop immediately after the injection, is of importance. From the amount of time that it takes for substances to diffuse through the eye (as shown by the previous experiments with potassium ferrocyanide already mentioned), it is evident *that the substances which had been introduced into the vitreous must have already been for some time in the region of the anterior chamber and corneo-scleral junction, and have been exerting their action there, before the attack of which that action was the cause, had taken place.*

The pathologico-anatomical condition found at this time was perfectly negative, apart from the œdema of the cornea and apart from the altered chemical composition of the aqueous and vitreous that was evident from the presence of the small clot in the anterior chamber and the clotting of

the vitreous. Nevertheless, the subsequent results of the experiments afford a clear explanation of the cause of the attack.

The first *visible* effects of the injections were processes of coagulation. Since the vitreous and aqueous under normal conditions do not coagulate even under the influence of the ordinary hardening agents, they must be mingled with an albuminous transudate, which under certain conditions may even undergo coagulation during life. But while the albuminous transudate derived from the vessels of the retina, the ciliary processes, etc., makes its way into the vitreous. Petit's canal, and the posterior and anterior chambers, the albuminous transudate derived from the vessels of the corneo-scleral junction passes into those interspaces of the tissue that serve for the channels of outflow from the eye. *If the transudate coagulates spontaneously, the channels of outflow are blocked up, and an attack of glaucoma is produced.* After some time the same process that is observed on a large scale in the aqueous takes place on a small scale in the interspaces of the tissues, *i. e.*, the coagulum contracts, and undergoes resorption. In this way room once more is made for the outflow, and the attack of glaucoma ceases. Indeed, it is probable that by the processes of exudation and coagulation the tissue-spaces are actually dilated and the outflow thus rendered freer than normal, since ordinarily after the glaucomatous attack a noticeable reduction of the tension below the normal takes place.

Now, by the blocking up of the anterior channels of outflow the passage of the injected irritants through the latter is cut off, and the action of these irritants temporarily ceases; but after the contraction of the coagulum, if there is any injected matter still present, the original processes may be repeated at the corneo-scleral junction and in the anterior chamber, so that in this way we may have the repeated formation of a lenticular exudate and repeated attacks of glaucoma.

The irritant action is manifested very soon also in the *immediate neighborhood of the injected matter*, and this latter, therefore, becomes encapsuled in a coagulation membrane,

and thus its further activity is prevented altogether or very much weakened. The exudate in the vitreous that forms this coagulum springs, to all appearances, primarily from the vessels of the retina. In this regard, there is an apparent contradiction between the results obtained by injecting the non-irritant potassium ferrocyanide and those got by injecting irritant substances, since in the former case a direct exit through the posterior channels of outflow (papilla and optic nerve) is generally impossible to demonstrate. But this is explained very readily by the fact that non-irritant solutions *simply pass through* the anterior channels of outflow, while the irritant solutions produce there an obstruction to their own passage, so that their path is deflected into other channels which, under ordinary circumstances, are but little, if at all, employed. In the latter case, therefore, the corneal margin and the optic-nerve entrance would act vicariously for each other.

The *subsequent course* depends largely upon the intensity of the irritation. If the latter is slight (*e. g.*, in case III.), there need not necessarily be any clinically-visible, spontaneously-coagulating, lenticular exudate formed in the anterior chamber. If the irritation is more violent, an exudate of this character develops very speedily, in some cases so speedily that no preliminary increase in tension can be made out (experiments with the "oleum cinereum"). Furthermore, in the course of about two days, if oleum cinereum is used, but later if the injections are made with dilute oil of turpentine, a cellular infiltration is produced in the vicinity of Schlemm's canal (and to a less extent in the retina and optic papilla), which causes disintegration of the tissues, and ultimately, if the irritation keeps on, develops into chronic iridocyclitis and iridochoroiditis. The fact that these last-named processes took their origin from the angle of the anterior chamber was very evident, from several experiments.

If, on the other hand, the irritation is sufficiently slight, it may happen, after the prodromal glaucoma has run its course, that everything becomes, apparently, normal once more, and remains so for a longer or shorter space of time,

until some fortuitous exciting cause produces new symptoms. In this connection, experiment III. is of extreme interest, since in the course of it two spontaneous attacks of glaucoma and two that were produced by atropine were observed. In my opinion, the second spontaneous attack was caused by spontaneous rupture of the membrane that encapsulated the drops—a rupture which could be demonstrated ophthalmoscopically, and as a result of which the remainder of the encapsulated irritant liquid was enabled to exert a renewed action. *After this had taken place, no subsequent attack could be produced, even by atropine.* The ultimate anatomical state was normal, especially in the region of the anterior channels of outflow, the only exceptions being the vitreous, which, being subjected to the direct action of the injections, had become completely shrunken, and the posterior cortex of the lens, which, being close to the point where the drops were injected, showed cataractous changes (Fig. 7).

There is quite a marked difference in the behavior of the eye after the injection of dilute oil of turpentine (cases IV.–VII.) and oleum cinereum (X.–XIII.). The former speedily produced a considerable elevation of tension. On the other hand, the spontaneous coagulation of the aqueous was but slightly pronounced. With the oleum cinereum, on the contrary, an abundant coagulating exudate formed in the anterior chamber, and cellular infiltration could be made out as early as the beginning of the second day. The action of the dilute oil of turpentine was at the outset limited almost entirely to the anterior channels of outflow, and the experiments with it, therefore, gave very well indeed the clinical picture of a prodromal attack of glaucoma. The action of the oleum cinereum was quicker, more widespread, and more violent, and hence better suited for demonstrating the anterior and posterior channels of outflow by the cellular infiltration it caused.

The anatomical conditions found after the action for a short time of stronger irritants (turpentine and olive oil 1:2) —namely, cellular infiltration of the vicinity of the angle of the anterior chamber and the optic papilla,—and also those

found after injecting oleum cinereum, correspond completely to the conditions found in *glaucoma imminens*, which I described in my first publication upon glaucoma and of which Nettleship afterwards published several cases (sarcoma of the choroid before the beginning of the glaucomatous symptoms). In connection with this anatomical state there is no increase of tension; in fact, according to Nettleship, there is actually a decrease of tension.

Nevertheless, in my experiments the subsequent course was not that of a glaucoma, since the action of the liquids used by me was obviously different in a qualitative way from that of the products of tissue-metamorphosis that are the efficient agents in human glaucoma. These latter products, which we may conjecture to exist in certain cases (sarcoma and glioma, a cataractous lens undergoing solution in the vitreous, certain detachments of the retina, etc.), obviously act less violently but in a permanent fashion, and thus *inter alia* produce in the angle of the anterior chamber an adhesive form of inflammation of a different kind from that caused by oil-of-turpentine injections or by the products of metamorphosis of the bacteria of suppuration. Perhaps repeated injections of very dilute oil of turpentine might have accomplished the same result.

For those who are not so prejudiced as to cling obstinately to preconceived ideas, the pathology of glaucoma will be clear from the results of these experiments and of the anatomical conditions found in the later stages. If we exclude glaucoma simplex, which runs its course without elevation of tension and without any inflammatory symptoms, and which is only an optic nerve atrophy with a large physiological excavation, a disease in fact that v. Graefe originally distinguished from real glaucoma under the name of "amaurosis with excavation of the optic nerve"—if, I say, we exclude glaucoma simplex, *glaucoma is an irido-cyclitis in which the anterior channels of outflow of the eye are occluded, temporarily at first, afterwards permanently;* the permanent occlusion being due to adhesion of the periphery of the iris to the cornea, *a condition which in the advanced stages is never absent.* Hence it is too that the symptom of

increase of intraocular tension plays such an essential part in all the varieties of true glaucoma, although it is by no means continually present, and may actually alternate with diminution of tension.

So long as the inflammatory irritation in the anterior outlets of the eye is a transient one, so that it is still possible for everything to return to its normal condition, only prodromal attacks occur, and the angle of the anterior chamber remains normal; and it is not until an anatomically demonstrable permanent obstacle to outflow is produced by the development of a peripheral anterior synechia that the real glaucomatous attacks set in. Then nothing but the making of a new channel of outflow from the anterior chamber into the subconjunctival tissue, which is effected by means of the peripheral section at the corneo-scleral junction, is able to produce a return of equilibrium in the conditions of tension in the eye. It does this in a great number of cases. Should, however, an obstacle to the outflow still exist—and this may possibly be due to the fact that the processes of coagulation in the channels of outflow still keep up,—we then get the *malignant* forms of glaucoma which generally run a very acute course.

Glaucomatous irido-cyclitis is distinguished from the ordinary irido-cyclitides by the fact that in the latter the nocuous element has from the outset its seat in the iris and ciliary processes, while in the former it circulates in the vitreous and aqueous and in its exit from the eye causes an inflammation of the iris and ciliary body starting from the region of the angle of the anterior chamber. It must be admitted that in the genuine glaucomas we can make no definite statement in regard to the nature of this element; while in the secondary glaucomas, *i.e.*, in those for which we know an exciting cause, whether it be a malignant tumor, a luxated lens, or retinal hemorrhages, we must assume the existence of such an element circulating in the vitreous.

The *injurious action of mydriatics* is explained by the fact that they cause hyperæmia of the fundus, and consequent acceleration of tissue-metamorphosis there and in the vitreous, so that the nocuous elements contained in the lat-

ter are hurried along faster and in greater quantity towards the channels of outflow of the eye. When the nocuous element no longer is present (*e. g.*, in experiment III. after all the oil of turpentine had been discharged), instillation of atropine fails any longer to produce a glaucomatous attack.

A few words in regard to the other anatomical conditions found in my experiments.

The *aqueous*, even in cases in which it did not undergo spontaneous coagulation, showed the formation of a coagulum in the hardened eye, a proof that it was pathologically altered in composition, and particularly that it contained albumin. The coagulum when formed spontaneously was in the early steps nearly devoid of cells; in the later stages it contained them in greater or less amount. The round cells obviously come in but very small part from the anterior surface of the iris. The larger portion lay mainly upon the anterior surface of the exudate directly in contact with Descemet's membrane; the greater amount being along the lower half of the cornea, and the cells being especially abundant in Fontana's space, giving the appearance as if they had been filtered off from the latter. They obviously originated mainly from the region of the posterior chamber.

Apart from one case (IV.) in which a similar cellular fibrinous coagulum was contained in the tissues of the rest of the iris, one (VII.) in which the anterior endothelium of the iris was partially detached, and one (XI.) in which there was œdema of the posterior epithelium, the *iris* was practically normal. Changes in the iris, therefore, cannot be the determining cause of the fibrinous character of the aqueous.

Where there has been increase of tension and for some time afterwards, thickening of the *cornea* is regularly found. This thickening is often obviously caused by œdema and for the most part is distributed very uniformly over the whole surface of the cornea. Only in a few cases could a delicate, vapor-like opacity of the centre of the cornea be made out by clinical observation. Since in the experiments the cornea remained absolutely intact, no assumption is possible except that after the occlusion of the channels of outflow at the corneal margin a certain quantity of liquid

still made its way by diffusion from the anterior chamber into the cornea, and when the obstruction was removed came out once more either from the anterior layers (towards the subconjunctival tissue) or the posterior layers (through the sclera) (*cf.* Figs. 2, 3, and 4). Thus my view, enunciated almost 20 years ago, that the endothelium of the posterior surface of the cornea forms no absolute bar to the passage of liquids, but merely regulates their passage, finds renewed confirmation.

It is not surprising that, after an encapsulated drop of irritating liquid has remained for some time in the vicinity of the posterior pole of the *lens*, lenticular opacities especially in the posterior cortex should occur. It is a matter of interest that in experiment II. punctate cataractous formations, could be observed in the anterior capsule also, it being well-known that anterior capsular cataract is a clinical evidence of a complicated cataract, *i. e.*, of one associated with pathological changes in the fundus.

The changes in the *vitreous*, apart from the coagulation membrane that formed about the injected substances, consisted essentially in an imbibition with a coagulable transudate, so that the entire vitreous became solid under the influence of hardening agents. In the earliest stages the coagulated vitreous was almost devoid of cells; later an infiltration with round cells developed, beginning in the most peripheral layers adjoining the retina and ciliary processes.

The material for the coagulation of the vitreous is in part that which is produced by the retina, and serves for the condensation of the hyaloid membranes. If the irritation is but slight and transient (experiment III.), the vitreous and hyaloid separate from the retina and may contract to very small dimensions without causing further changes in the latter. The place of the vitreous is then occupied by liquid.

If the irritation is more intense, it makes itself felt upon the *retina* even before the shrinking of the vitreous. The innermost layers of the retina are infiltrated with round cells, which originate in the most peripheral layers of the vitreous, and are generally most abundant about and upon the optic papilla. When the irritation lasts a long time,

total detachment of the retina occurs (experiments I. and IX.). This, however, does not owe its development simply to the infiltration and subsequent retraction of the inner layers of the retina. Its beginnings are manifested at a very early period, before there can be any idea of such infiltration and retraction taking place, in the vicinity of the optic papilla, where folding of the retina and the presence of a coagulated exudate between these folds and the choroid (cases XI., XII., XIII.) prove the existence of a commencing detachment of the retina.

This concentration of the exudate about and upon the *papilla* compels the conclusion that channels of outflow are present in the interstitial tissues of the optic papilla, and that similar processes take place in the latter as in the tissues of the anterior channels of outflow. The accumulation of liquid about the papilla between the retina and choroid would hence be originally a symptom of engorgement. This is the spot in which the halo is found when there is glaucomatous excavation. In our cases a total detachment of the retina developed out of these circumpapillary changes; in glaucoma an atrophic spot, resulting from an adhesive inflammation, forms in the choroid; hence the so-called halo.

While evidences of inflammation were found in the retina and papilla, these evidences did not extend any great distance behind the lamina cribrosa. The outflow from this point therefore appears to take place not so much in the optic nerve itself as in the tissues surrounding the central vessels and in the spaces ensheathing the nerve.

The *choroid*, in the days immediately succeeding the injection, showed no visible changes.

In conclusion, the results of my researches may be briefly recapitulated as follows:

The experiments had for their object (1) to *render visible the channels of outflow from the eye (especially those of the anterior chamber) by means of the inflammatory changes which aseptic agents provocative of inflammation, that had been introduced into the vitreous, would excite* in the course of

their passage through these channels. The results agreed with my former researches with indifferent substances, inasmuch as they both showed that the outflow (which does not occur in closed cavities, but in the interspaces of the tissues), takes place mainly from the angle of the anterior chamber through the sclera to Tenon's capsule, and to a less extent through the unchanged cornea into the subconjunctival tissue. But since, as a result of the changes at the corneo-scleral margin, an occlusion, although but a temporary one, took place in the anterior channels of outflow, a further result occurred in these cases which did not occur when indifferent substances were used, and which consisted in the fact that the existence of the posterior channels of outflow was likewise made evident by means of inflammatory processes taking place in them. Of course, it is understood that this holds good for the passages of outflow only for liquid which has got into the vitreous chamber, and not for that which comes from the tissue of the choroid, for example. The starting-point of these posterior channels of outflow proved to be, as it is generally supposed to be, the optic papilla, and especially its interstitial tissue.

2. It was the object of the experiments to *produce inflammatory processes of an adhesive nature in the anterior channels of outflow*, and produce consequently *artificial glaucoma* by means of the inflammatory agents as they passed out through these channels. This was only partially successful. Only a temporary occlusion of these channels, in other words, only prodromal glaucomatous attacks, could thus be produced. The subsequent course either was that the inflammatory agent gradually disappeared and thus a cure was produced, or that the agent acted differently from the way in which it had been anticipated that it would act; in fact, cellular infiltration, and not a simple adhesive inflammation, set in in the spots affected. A *continuous occlusion* of the anterior channels of outflow, such as is characteristic of fully developed glaucoma, could not be secured.

Nevertheless the results of the experiments are of the greatest importance for the pathogeny of glaucoma. For the first time the attempt was made deliberately and success

fully to produce what are clinically speaking prodromal attacks of glaucoma, and what from the point of view of anatomy is an isolated inflammation of those spots, namely, of the vicinity of the *sinus of the anterior chamber and the optic nerve*, which in uncomplicated glaucoma are likewise the only ones that show pathological changes—to produce in short the picture of glaucoma imminens, as I have already described it in man, consisting in cellular infiltration of these two regions occurring before the slightest clinical symptoms were yet present. Nettleship described a similar picture occurring in the case of eyes with sarcoma of the choroid before the development of glaucomatous symptoms. Obviously, in order to ensure the development of a full-fledged glaucoma, the inflammatory agent *must act continuously, but less intensely* than did the material that I used, *i.e.*, must probably act like the substance secreted by a growing malignant tumor. Whoever succeeds in finding such a continuously acting inflammatory agent will have it in his power, by introducing this substance into the vitreous, to produce a fully developed glaucoma. This seems, however, scarcely necessary, since every sarcoma of the choroid, every lens displaced into the vitreous by couching is really fully equivalent to an investigation of this sort, and could not possibly be made more free from extraneous influences if it were done experimentally.

It is hoped that with the results here given the theory of the neurotic nature of glaucoma has been done away with once for all. An agreement upon this point could never have been reached as long as the confusion was maintained between things so distinct clinically and anatomically as the so-called glaucoma simplex and the inflammatory forms of glaucoma. A. v. Graefe's clinical acuity first recognized their non-identity, and the results of subsequent anatomical investigations have more and more confirmed his view. If we call *glaucoma simplex* what it really is, *i.e.*, *optic-nerve atrophy with excavation*, and the *true forms of glaucoma* we call *irido-cyclitis anterior* (*i.e.*, a form in which the exudate is found mainly on the anterior aspect of the parts), an inflammation which may occur in every possible degree of acuity

and intensity, we then have designations in which the clinical symptoms and the anatomical conditions are strictly correlative; in short, we have well known and readily recognizable clinical pictures representative of diseases that have a real existence.

EXPLANATION OF THE ILLUSTRATIONS.

FIG. 1–4.—Different successive stages of the passage through the anterior channels of outflow in the eye of potassium ferrocyanide that has been introduced into the vitreous.

FIG. 5.—Gelatinous exudate in the anterior chamber in experiment I., 4 days after the injection of oil of turpentine into the vitreous.

FIG. 6.—Beginning of anterior capsular cataract in experiment II., 3½ months after the introduction of the oil of turpentine into the vitreous. In this case on the fourth day a plastic hemorrhagic irido-cyclitis had developed as a result of traumatism.

FIG. 7.—Section of eye No. III., 4½ months after the introduction of oil of turpentine diluted with olive oil.

CONTRIBUTIONS TO THE CONGENITAL ANOMALIES OF THE FUNDUS OCULI.

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Translated (abridged) by Dr. J. A. Spalding, Portland, Me.

(With two chromo-lithographic Plates, X. and XI. of Vol. XXVIII. Germ. Ed.)

I.—TWO CASES OF PARAMACULAR COLOBOMA OF THE CHOROID, WITH TUBULAR FORMATIONS.

HIRSCHBERG has shown (*Berlin. klin. Wochensch.*, 1892, p. 327) that cylindrical formations in the vitreous are often mistaken for encapsulated cysticerci, and even appear as such in literature, amongst which cases he cites those by Liebreich and v. Graefe (*v. Graefe's Archiv*, i., p. 348, and ii., p. 263), to which may be added that by Teale (*R. L. O. H. Reports*, vol. v., p. 310). In the May number of last year's (1894) *Centralblatt*, he returns to the subject, and proves that the first two cases must be connective-tissue formations in the vitreous.

Hirschberg was also the first to dissipate the myth of the spontaneous cure of cysticercus of the interior of the eye, and to place the diagnostic error where it belongs. He communicates in the same paper an additional case of a congenital formation in the vitreous produced by the persistence and condensation of the connective tissue surrounding the hyaloid artery. His chief diagnostic points are: Discoloration of the iris; a bluish cylinder which extends from near the papilla through the vitreous, is fixed

behind by tent-like fibres, and terminates in front in minute fibres and membranes which almost reach the posterior capsule of the lens; and, finally, loss of vision in the affected eye, with a dark spot in the field of vision, and strabismus.

I will now describe two similar cases, which vary somewhat from those so far described, and which I have had a chance of watching for many years.

CASE 1.—Mr. A., aged twenty-four, has always had a defective left eye, which deviates outward 40°.

The iris is of the same color as the other.

Media transparent, papilla very white, atrophic, excavated, slightly pigmented, and exhibiting a conus (Fig. 1). The upper temporal quadrant shows a congenital defect, which seems to be a paramacular coloboma of the choroid, and of peculiar form, as shown in the figure. The entire coloboma is devoid of choroidal vessels, but contains retinal vessels, and is well defined from the remaining fundus of the eye, which is normal in color.

The angle between the two portions of the coloboma shows a cylindrical structure, with one end in the retina, and the other, larger, extending into the vitreous. It is about the shape of an almond, is about half the diameter of the papilla, and near the retina gives off an offshoot, but as it extends forwards has no branches. There is no trace of vessels, but slight pigment around the place of attachment. The refraction of the retina at the point of implantation is + 1 D. S., and that of the summit of the growth + 6 D. S.

The retinal vessels show but few changes, and these can be studied in the sketch. The chief rarity is that the inferior retinal artery arises from the upper part of the disc, and bends downward properly after a short excursion.

Vision $\frac{1}{3}\frac{0}{0}$, and field concentrically contracted.

I have followed the case for years, and there has never been any change in the ophthalmoscopic image of the eyes.

CASE 2.—The second case is that of a girl of fifteen, who has a bridged-over coloboma of the iris of the right eye downward, so that she has a double pupil. Ah of + 2 D., and a normal fundus oculi. Vision $\frac{6}{1}\frac{0}{0}$.

The left eye diverges 20°, but the iris is of the same color as that of the other eye. The optic papilla (Fig. 2) in the erect image is oval, and covered with a stripe of whitish-gray tissue,

which begins in the porus of the papilla, covers the exit of several of the retinal vessels, and extends toward the periphery of the fundus of the eye, decreasing in diameter at first, and then enlarging as it enters upon a defect in the choroid of a figure-of-8 shape. The choroidal coloboma offers attachment for a greenish-gray cylindrical formation, which then extends into the vitreous. Refraction of papilla + 1 D. S.; that of the coloboma + 3 D. S.; of the body of the neoplastic structure about + 8, and the extreme summit + 10 D. S.

Vision $\frac{6}{60}$, and field defective on nasal side.

I will not attempt to establish any hypothesis concerning this structure, but I think that it is like those described by Hirschberg and Dimmer,[1] because they, as well as v. Graefe and Teale, speak of similar structures in the vitreous. In all of these cases, however, the neoplasm arose from the papilla, so that they must have owed their existence to the hyaloid artery and its sheath. But in both of my patients it arose *at a distance from the papilla*, amidst defects in the choroid, so that it would seem a new sort of congenital defect.

It should also be emphasized, in opposition to Hirschberg, that both irides were of the same color; the outer end did not terminate in an awning-shaped expanse, and there was no central scotoma, only peripheral contraction of the visual field.

II.—A CASE OF CHOROIDAL DEFECT, WITH TENT-LIKE PSEUDO-MEMBRANE IN THE FUNDUS OF THE EYE, TOGETHER WITH MALFORMATION OF THE PAPILLA.

Miss L., aged eleven, has divergent squint of O. D., beginning two years ago, and subsequent loss of sight. The left eye is normal.

The right eye has vitreous opacities, and a curious ophthalmoscopic image (Fig. 3). There is no papilla, but at its situation begins a yellowish nine-pin-like structure lying horizontally, with a blunt, round, small end at the papilla, and then extending out to and across the macula to the temporal periphery of the eye. Whatever remains of the original papilla is embraced by the small

[1] These ARCHIVES, vol. xiv., p. 72.

end of the neoplasm, because the retinal vessels originate at that spot alone. Attention should be drawn to the difference of level between the horizontal structure and the rest of the retina, the refraction of the former being + 4 D. S., while that of the retina is barely + 1 D. S.

We next look at the shiny white tissue lying in front of the horizontal structure, and terminating in a tent-like appearance, which is a rarity in ophthalmoscopic images. It seems to be attached to the middle of the nine-pin-shaped structure, and towards the termination in a tent shape, projects well into the vitreous and toward the equator of the globe. The extreme fibres spread apart, and permit a clear view of the fundus of the eye.

Coursing down from this white patch is a shiny line extending farther along into a slight serous detachment of the retina. Directly above the tent-shaped termination is a defect in the choroid, seemingly connected with the horizontal structure, but proved by the refraction ophthalmoscope to be an entirely different structure—a sort of coloboma of the choroid.

Finally, we observe the ring of brownish pigment surrounding in its embrace the nine-pin-shaped body, the coloboma of the choroid, and part of the whitish shiny tissue with the tent-like termination.

There can be no doubt, that the nine-pin-shaped figure is formed by the retina, mechanically raised from its choroidal foundation, and that the cause of the whole is the fibrous matter, which in its turn must be regarded as the sequence of pathological processes in the fœtal connective tissue of the secondary ocular vesicle. The fibrous substance originated in the region of the papilla, and in the course of growth of the eye produced traction upon the papilla and the retina. At the age of ten, vitreous fluidity was excited by unknown means, together with a serous retinal detachment in the inferior quadrant of the retina, and this was what first called attention to the pathological condition of the eye.

III.—A TYPICAL CASE OF PERSISTENT HYALOID ARTERY.

Miss K., æt. thirty, has never seen well with O. D., and three years ago suffered from papillitis of O. S., with vision reduced for many weeks to $\frac{1}{4}$, followed by gradual restoration of the normal conditions and of good sight.

O. D. externally normal, and without deviation. Slight calcareous formation in periphery of lens. A whitish strand begins near the rear of the lens, and extends to the papilla, where it spreads out into a globular formation, which cannot be well seen unless the eye is turned up and in, for it runs so directly from the lens to the papilla that its course cannot otherwise be followed.

Examination in the erect image (Fig. 4) shows a well-formed papilla with much pigment on one side, and on its lower half the greenish termination of the hyaloid artery. The rest of the fundus seems normal. Myopic astigmatism -3 D. C. $\frown -2$ D. S. The refraction of the globule is about -1 D. S., and the summit of the cord is best seen with $+11$ D. S. Vision with best lens, $\frac{1}{3}$. Vision of O. D. normal with lens.

The case needs no commentary.

A CONTRIBUTION TO THE OCCURRENCE AND ORIGIN OF CONGENITAL ANOMALIES OF THE EYES.

By Dr. HUGO WINTERSTEINER, of Vienna.

Translated (abridged) by Dr. J. A. Spalding, Portland, Me.

(*With one chromo-lithographic Plate XII. of Vol. XXVIII. of Germ. Ed.*)

OUR knowledge of the developmental history of the eye is still incomplete, partly owing to the obstacles raised by the nature of the material to be examined, and partly to the difficulty of deciding what the specimens actually mean. There is, however, a way in which we can enlarge our knowledge of these processes, and that is by observing and analyzing congenital malformations of the eyes, for they offer new keys to the anatomical relations of the fœtal organ, or they confirm the experiences of histological studies, or if the latter cannot be brought into unison with one another, they excite us to new researches. Hence every new case of congenital anomaly which throws any light on our present knowledge, or confirms the same, excites our immediate interest. For all these reasons, I beg leave to report the following case:

Adolph W., æt. thirteen, suffered from inflammation of the eyes when three years old, and was treated for a year. His eyesight has always been poor, and he has not been sent to school. Two years ago he observed that the left eye was the better of the two, and as the right grew worse he came for an opinion, February, 1893.

Congenital epicanthus, no strabismus, both corneæ very hazy,

iris and pupils normal. After dilating the right pupil, the following changes were seen :

Simple illumination shows an opaque, glittering mass in the pupillary field, with cords extending above and below (Fig. 1).

The outer cord lying in front of the other gives off a second at right angles, which extends backward to the retina, branching as it passes along. Most of the branches are so tense that, on moving the eye to and fro, no motion in them is perceived. Finally (Fig. 2), there are two horizontally situated, grayish sort of furrows, which extend backward and are inserted into the lower margin of the papilla. The one running toward the nose carries an enlarged vein on its surface, whilst the other has a vessel so small that whether a vein or artery cannot be determined.

The papilla is very white, with an ill-defined border, but is not swollen or excavated. Its vessels are nearly normal. A few patches of pigment are scattered about it. The lower vein passes out a short distance, then turns sharply above the level of the retina, and sinks back again. At one spot it lies close to the neoplastic tissue in the vitreous.

The fundus is normal, only there is no trace of a macula. Near the upper margin of the papilla is a white streak, about the size of a vessel, which passes off into the periphery without much change. It is bordered above in part of its course by a line of pigment. This streak is actually behind the retina.

The upper, inner quadrant of the retina is marked by a similar whitish streak, which is, however, much more irregular in outline.

The globular structures in the vitreous are well depicted in the figure and need no further description. Owing to the haziness of the cornea and the poor control of the eye, the refraction of these masses could not be exactly determined.

Fingers were counted at 2 *m.*, O. D., excentrically. The field could not be accurately tested, but an offhand test showed nearly total loss in the upper half. Vision O. S., $\frac{1}{10}$, and Jaeger 3 at 16 *cm.*, but no improvement by glasses, owing to irregular astigmatism.

Here, then, we have a globular connective-tissue opacity in the vitreous, extending toward the periphery of the globe, retina, and papilla, in the form of connective-tissue bands and cords. The papilla is white, the vessels dislocated, and the retina interspersed with white striæ and pigment.

This condition of affairs may be congenital or due to dis-

turbances of development, or to intraocular hemorrhages or similar conditions.

Inflammation may be excluded, in my opinion, because a part of the alterations—the epicanthus, the abnormal vessels, and the tortuous vein—will not fit into such an explanation. Moreover, the vitreous changes are not such as we are accustomed to regard as inflammatory in origin, for inflammatory products so abundant as to produce such globules and cords are invariably associated with irido-cyclitis, deposits on the capsule, and vitreous opacities, of which there are no traces here. The only possible inflammation may be the so-called retinitis proliferans, in which, as the result of hemorrhage, we see connective-tissue neoplasms in the vitreous and retina. But in all of the cases of this disease which I can recall, the ophthalmoscopic image varied completely from that in the case now under observation.

Hence it seems that the condition here depicted is either a congenital anomaly or defective development, or intrauterine inflammation, and this view I will try to substantiate by the symptoms.

The vascular abnormities must be congenital or due to defective development of the vessels, because they cannot be explained by subsequent obliteration or thrombosis or secondary enlargement, since their origin from the papilla is abnormal. More than that, the absence of a macula, and the passage of a large vessel through its locality, shows a congenital defect.

Mention must also be made of the vein which, coming from the periphery of the retina, arises just below the macula region above the level of the retina, pushes into the vitreous, and then, bending in hook-shape, returns parallel to its former course in the retina, and then passes to the macular region and papilla. The only cases of vascular formation accessible to me were in *front of the papilla* and *arterial*. The only case resembling mine is one by Hirschberg,[1] in which the retinal veins formed a vascular network on the papilla and partly to one side, but these did not press forward into the vitreous, but lay on a level with the retina.

[1] *Centralbl. f. Augenhlkde.*, 1883, p. 289. Note to a paper by Czermak on vascular formations in the vitreous.

Czermak's opinion, that these are the remnants of an embryonal vitreous arcade, originating from anastomosis and ramification of the embryonal vitreous vessels, seems to be appropriate also to the appearances in my patient.

The appearances in the vitreous are congenital, though what they mean is doubtful, as I find nothing analogous in the literature at my command. Those most resembling it have been described by Eversbusch[1] and Dimmer,[2] though theirs were smaller, consisting in a tent-like detachment of the retina, extending into a connective-tissue prolongation to the posterior capsule of the lens. Vessels were given off to the retina. Eversbusch thought that the formation reached the capsule, whilst Dimmer was of the opinion that a former junction had been destroyed. Both authors speak of the condition as being a remnant of the hyaloid artery.

Beside being sure that my case is congenital, I also insist that it is not a persistent hyaloid, or a Cloquet's canal. For these never appear simultaneously, and I have never heard of a Cloquet's canal with vessels, to say nothing of veins, on its surface. Ricchiardi has, however, discovered[3] projecting veins in the vitreous, up to the third month of fœtal life, and there was distinct anstomosis between the capsular artery, and the peripheral vessels of the vitreous.

We can therefore imagine that, owing to undue influences, these fœtal vessels did not retrogress as usual, that their walls proliferated, and were eventually enveloped in a connective-tissue mantle, as shown in the figures. The arteries can be explained in the same way as we explain their appearance in neoplasms in the vitreous.

Jaeger was the first to call attention to cords in the retina,[4] but since then only few similar cases have been depicted (chiefly by Perrin, Hirschberg, and Magnus), and all entitled retinitis striata. Other cases so far published refer to similar images, but to dissimilar processes, as is plain to any careful observer.

The nature of these striæ is still in the dark, because no one has yet been able to watch their origin or growth.

[1] *Mittheilungen aus d. Univ. Augenklinik. z. Muenchen*, 1882.
[2] These ARCHIVES, vol. xiv., p. 73.
[3] Nagel's *Jahresbericht*, 1872, p. 73.
[4] *Atlas*, 1869.

Similar appearances are seen after detachment of the retina, and Liebreich[1] has pronounced them to be coagulates, precipitated from subretinal effusions, whilst Dimmer looks at them as developments of connective tissue in the retina, and Braun,[2] as atrophy of the pigment epithelium.[3]

Stellwag saw white striæ in the retina after retinitis albuminurica, and Berger in eyes with normal vision. The latter thinks that they are the residue of fœtal neuro-retinitis, or of a retinitis occurring soon after birth. The titles of retino-choroiditis and retinitis given by other authors, indicate that they have inflammatory alterations in mind.

Finally, Speiser[4] urges that the retrovascular striæ originate in hemorrhages in the external layers of the retina.

I would also advance another possibility. The striæ are generally arranged radiatingly, and they seem as if passing over elevations in the retina. If now we recall the developmental history of the eye, we remember that the retina in its development often lies in folds, of which we see abundant traces in the fœtal eye as well as in the new-born. It is only when the globe has increased in size with the growth of the child, that these retinal folds disappear. The idea therefore suggests itself, that all the retinal striæ so far observed have some unknown connection with the embryonal folds of the retina.

Other arguments in favor of this origin of the striæ is that no one has so far seen their beginning or development. Onisi alone,[5] after looking at a patient several months later, saw striæ that he thought he had not seen before, but he would not say that they may not have been present at the first examination. Neither have any inflammatory symptoms ever been reported by the patients themselves. They accidentally discovered loss of sight in one eye. Additionally in my case, the striæ, the abnormal vascular distribution, and vitreous opacities insist on a congenital explanation. Finally, Berger and Michel report similar cases, in which the conditions must have originated at an extremely early age, even if they were not congenital.

[1] *V. Graefe's Archiv*, ii., p. 256.
[2] *Beitraege z. Augenhlkde.*, 1893, Heft, xii., p. 30.
[3] See also a paper in this number of these ARCHIVES by Dr. W. A. Holden.
[4] *Beitraege z. Augenhlkde.*, Von Millinger, Basel, 1893.
[5] *Mittheilungen a. d. Augenklinik. Tuebingen*, ii., 3, p. 377.

A CASE OF DOUBLE COLOBOMA OF THE CHOROID.

By C. A. VEASEY, M.D., Philadelphia, Penn.

(*With an ophthalmoscopic drawing in the text.*)

THE following case of coloboma of the choroid, which seems to possess some points of interest peculiar to itself, presented the following history:

A. S., female, aged twenty-four, came to me for the treatment of a variety of symptoms which she attributed to her eyes. For several years she had had frequent frontal and occipital headaches accompanied by nausea, and occasionally by vomiting, always aggravated by doing any near work. The headache was very severe, and the patient stated that almost every week she was obliged to spend one day in bed on account of it—usually a day in the latter part of the week. There were also present symptoms of accommodative asthenopia and ciliary pain. She had been wearing sphero-cylindrical lenses for three years, with relief of the symptoms for one year after they were obtained. The family history was good. Both her parents and herself denied having had any specific lesion. The general health was very good excepting the disturbance arising from the ocular symptoms. The vision and accommodative power were as follows:

O.D.V. $\frac{5}{7.5}$ Jäger, No. 2——3″–28″

O.S.V. $\frac{5}{15}$ Jäger, No. 2——5″–10″

After she obtained her correction, which was as follows;

O.D. + S. .75 D ⌒ + C. .75 D. ax. 90°

O.S. + S. 1.25 D. ⌒ + C. 1.75 D. ax. 90°

the vision was for the right eye $\frac{5}{5}$ full, for the left eye $\frac{5}{5}$ partial, the best working distance being 16″.

The tests for muscular balance showed no trouble in this direction. Both pupils were normal, reacting quickly to light, convergence, and accommodation. The tension was also normal and the ophthalmoscopic changes were as follows :

O.D.—Media clear, disc oval, deep central physiological cup, absorbing conus to the nasal side of disc, cilio-retinal vessel to the temporal side about the junction of the upper and lower quadrants, choroidal ring all around, no macular disturbance.

O.S.—Media clear, oval disc, deep central physiological cup, choroidal ring all around, veins slightly fuller than normal, but blood apparently not darker than usual. About the distance of two thirds of the diameter of the disc below it and slightly to the nasal side, is a small circular coloboma of the choroid, greenish-white in color, which is about two thirds as large as the disc itself. The margin is pigmented all around while the bottom shows numerous elevations and depressions, presenting an opalescent appearance, and having on its surface two scleral vessels, the one to the temporal side sending off a branch. The depth of the coloboma varies from 1 to 2.50 D. in different parts. Passing over it from above downward is the inferior nasal artery. The surrounding choroid is in a perfectly healthy condition.

About the distance of two diameters of the disc below the coloboma just described is a second, oval in shape and much larger than the former, measuring, as nearly as could be ascertained, about $1\frac{1}{2} \times 2\frac{1}{2}$ diameters of the disc and being slightly narrower in the lower than in the upper half. The margin of this also is pigmented, much more heavily in the upper than in any other part, and the color, as in the first, is of a dirty greenish-white. The coloboma, in the upper outer portion and in the lower inner portion, slopes gradually from the margin towards the centre, while in the other parts the edge is more abrupt. The bottom presents numerous small elevations and depressions and has an opalescent appearance like the first, the depth varying from 2 to 5 D. The inferior nasal artery that passes over the upper coloboma from above downward continues its downward course, passing beneath a branch of the inferior temporal vein and crossing the lower coloboma in a curve, sending off a branch just before it reaches the lower margin. The branch of the inferior temporal vein just mentioned passes over the edge of the coloboma above, dipping beneath it so as to be entirely lost from view, and then, reappearing near the middle of the outer edge, passes downward and outward. Coming from beneath the upper

outer margin is a scleral vessel which sends two branches downward, one to the temporal and one to the nasal side, both being lost under the margin. Above and to the nasal side of the lower coloboma the choroid is distinctly thinner, though in all other directions it appears to be in a healthy condition. The accompanying illustration made from a water-color sketch by Mr. T. C. Lander of this city, shows very well the condition of the fundus.

The field of vision shows a scotoma for form and colors of the exact shape of the smaller coloboma, the larger one being so far in the periphery that it is indicated in the field only by a curve in the upper edge.

The defects are believed to be congenital, an examination by my friend, Dr. Edward Jackson, over three years ago revealing practically the same condition. No other congenital defects could be found in the patient, and an examination of the father and a sister showed nothing more abnormal than low refractive errors.

The case presents the following points of interest:

There are two separate colobomata of the choroid with a wide area of intervening healthy choroidal tissue, in which the field of vision is perfectly normal. Cases have been recorded in which a large coloboma was divided into different parts, as, for example, the case of Hirschberg, quoted by Bock[1] in his excellent monograph, where the coloboma was divided into three parts, while in the middle ran a thin strip of choroidal tissue; or Hoffman's[2] case, in which there was an intervening space of healthy choroidal tissue, but which was no wider than two thirds the diameter of the small coloboma, the latter being about half the size of the disc; but the author has been unable to find any case recorded in which the colobomata were so widely separated, and in which so much of the intervening choroid was in a healthy condition—in the present case the whole space, with the exception of a slight thinning above the lower coloboma, being normal.

The shape of the defects is of interest, the upper being circular and the lower oval. The visual field shows a scotoma corresponding in shape to the upper coloboma, and a curve in its upper margin corresponding to the margin of the lower coloboma. This agrees with the observations of Schmidt-Rimpler[3] and De Wecker,[4] though Johnson[5] claims that it is very seldom that the field shows a defect even slightly resembling the outline of the defect in the choroid.

It is also of interest to note that the choroid below the lower coloboma is in a normal condition. The defect is supposed to be due to the non-closure of the ocular fissure, but why it should have closed in certain parts and not in others, or why, if it were entirely closed, it separated above and below, leaving so wide a space perfectly healthy in almost the entire extent, is only a matter of conjecture.

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[1] Bock, *Die Angeborenen Kolobome des Augapfels*, p. 95.
[2] *Graefe-Saemisch Augenheilkunde*, p. 73
[3] Schmidt-Rimpler, *Graefe's Arch.*, xxiii, 4, p. 176.
[4] De Wecker et Landolt, *Traité Complet. d'Oph.*, vol. ii., p. 515.
[5] Johnson, *Arch. of Oph.*, 1890, p. 28.

DIABETIC RETINITIS.

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ALTHOUGH the existence of diabetic retinitis has been known for a long time, the literature on the subject is very meagre and unsatisfactory. The text-books on general medicine many of them do not mention the possibility of its occurrence, and the works upon the eye only give short statements that it may exist, but no satisfactory explanation of its symptoms. Having seen three cases of the disease within about a year, I think the scattered condition of the literature is apology enough for gathering the histories of all the cases of pure diabetic retinitis reported in literature and gleaning from them the characteristics, if there be any, that are indicative of the disease.

Diabetes being an infrequent disease and retinitis one of its less common complications, we have only a few cases reported, as diabetes is frequently complicated in its later stages (when retinitis most frequently occurs) by albuminuria. This has been considered by some as the cause for the retinitis. For this reason I have excluded from the summary all such cases, except one in which a thorough post-mortem examination had been made, and one of my own cases.

The more frequent complications, as weakness of accommodation, transient amblyopia, and cataract, had been known long before Edward Jaeger published the first report of a case of diabetic retinitis in 1856, a representation of which

is given in his ophthalmoscopic atlas (Fig. 64). This was followed by two cases reported by Desmarres in 1858. Although plenty of sugar but no albumen was found in the urine in these cases, the view was still held that possibly there had been a previous albuminuria which was absent at the time of examination. That diabetes alone could cause a retinitis was subsequently shown by H. D. Noyes in 1869, who carefully followed the history of a case of diabetic retinitis and found that while the eye symptoms bore a close resemblance to an albuminuric retinitis, there was no albumen present at any time in the urine, but abundance of sugar, and that the normal amount of urea was present.

Leber was able to collect the reports of 19 cases of diabetic retinitis in 1875 including one of his own. The histories of many of these cases were very meagre so that the presence or absence of albumen was only mentioned in 8 cases. In 4 of these there was no albumen, and they have been included in my report. His classical articles in *Graefe's Archiv*, vol. xxi., and the *Graefe-Saemisch Handbuch der Augenheilkunde* have been long considered authority on the subject. Hirschberg reported 24 cases in his articles in the *Deutsche klin. Woch.*, 1887 and 1890, 15 of which were pure diabetes. Lagrange reports from the eye clinic in Bordeaux the histories of 17 cases, 11 being in pure diabetes. Besides these more extensive articles Nettleship and others have reported cases which have been included in this report.

In order to get the ideas of different authors upon the subject, we will briefly consider them here.

Noyes (*Text-Book of Eye Diseases*, 1894), says: "Retinitis glycosurica is much less frequent than retinitis albuminurica. The features of both are to a great extent identical. The bright yellow patches are less liable to affect the macula; perhaps they lie more frequently between the disc and the macula. They are more nodular and irregular than in the albuminuric form." He then gives a description of the case reported in 1869.

Berry (*Text-Book of Ophth.*, 1889) states: "Retinitis occurs in some cases of diabetes, mostly those of severe type. The

form is not characteristic; it resembles albuminuric retinitis, but is accompanied by fewer changes in the region of the macula. Opacities in the vitreous are frequent."

DeWecker & Masselon (*Manuel d'Ophtal.*, 1889,) say: "Most cases of retinitis said to be diabetic occur in diabetic albuminuria; in reality true diabetic retinitis is very rare. Diabetic retinitis consists of simple hemorrhages alone or mixed with foci of degeneration; it is sometimes accompanied by large hemorrhages into the vitreous or coats of the optic nerve."

Juler (*Handbook of Ophthal. Science*, 1893) says: "Diabetic retinitis is very rare, and is so similar to the albuminuric form that it is almost impossible to distinguish the one from the other by means of the ophthalmoscope alone. The white spots in the macular region are large and irregular in outline in this affection, and they have no tendency to be radially arranged around the yellow spot. Hemorrhages occur in all parts of the fundus; the extravasations appear either in the form of dots or as irregular patches and lines. In the early stage papillitis is not a marked sign, but later on it becomes very prominent. The disease tends to progress; new vascular formations appear in the vitreous, and the latter becomes crowded with membranes and other opacities; lastly the retina becomes detached, with complete loss of vision. The retinitis is symmetrical."

Fuchs (*Lehrbuch der Augenheil.*, 1889) only mentions the disease to say that it is very rare, and that the changes in the retina are due to the altered condition of the blood.

Leber, in summing up his article in *Graefe's Arch.* (vol. xxi.), says: "The form of the retinal changes offers little that is characteristic by which we could judge that diabetes is the cause of the trouble. In many cases there are only retinal hemorrhages to which whitish spots of degeneration are added later, frequently accompanied by vitreous opacities. In other cases there is hemorrhagic retinitis as in disease of the heart or blood-vessels, in others whitish spots and hemorrhages which cannot be distinguished from albuminuric retinitis. The spots are small ($\frac{1}{4}$–$\frac{1}{3}$ O D, sometimes 2 O D), never run together, and are scattered over a large part of the fundus."

Schweigger says that all forms of eye disease are seen with diabetes, but none that bear the same relation to it, as for example retinitis albuminurica to Bright's disease or retinitis syphilitica to lues.

In opposition to this view is that of Hirschberg, who has grouped the cases of diabetic retinitis into three principal classes.

1. A characteristic inflammation of the centre of the retina with small bright spots and usually with small hemorrhages (retinitis centralis punctata diabetica).

2. Hemorrhages in the retina with the following inflammatory changes and degeneration (ret. hemorrhagica diabetica).

3. Rare forms of retinal inflammation and degeneration, the connection of which with the fundamental disease must be more fully proved. (He has seen three cases of retinal pigmentation in diabetes with contraction of the visual field and night blindness similar to retinitis pigmentosa.)

The first form he considers indicative of diabetes, and believes it is easily diagnosed from albuminuric retinitis which it most closely resembles.

Lagrange, reporting the cases from the ophthalmic clinic at Bordeaux, says of the disease: "Diabetic retinitis nearly always presents hemorrhages which may be large or small. It is usually easy to distinguish from the cardiac and syphilitic forms, but not so from the albuminuric." He then gives the table of differential diagnosis between the two diseases prepared by Badal, and which will be given below.

Its frequency cannot even approximately be determined as there are no incident statistics. In the ophthalmic clinic at Bordeaux among 20,000 eye cases there were 52 cases of diabetes. Of this number 17 had retinitis, 11 being in pure diabetes, and included in my list.

Galezowoski reported 27 cases with retinal changes, among 144 diabetic eye cases (19 per cent.), but gave no detailed description of them. This was much less than the number of cataracts of which there were 46 (31 per cent.). Hirschberg thinks that eye changes due to diabetes are much more frequent than is generally supposed, and says that retinal changes are probably present in all cases of long standing.

The age at which diabetic retinitis occurs is generally an advanced one, most of the cases occurring after forty-five years of age. The youngest case, that reported by Culbertson, aged six years, was an exception, as the diabetes was secondary to malaria, which may have produced the grave changes in the blood necessary to the production of the retinitis. The reason for its occurring at such an advanced age may be due to the changes normally occurring at this time of life in the blood-vessels, but it is more likely due to the fact that the disease takes a much more protracted course at an advanced age, sometimes lasting for a great many years. Retinitis only occurring at a late stage of the disease would be more apt to occur in such cases where the changes in the blood-vessels had an opportunity to develop.

As to the form of the retinal changes present, we have several types. The one most frequently met with is that described by Hirschberg as retinitis centralis punctata diabetica. The changes are somewhat differently described by the various authors, but they agree in all the essential points. Lagrange described some of the cases under the name of retino-choroiditis, but expressly stated that there were no pigmentary changes.

The changes consisted of small bright glistening spots of degeneration interspersed with minute hemorrhages principally in the central part of the retina. The spots are usually small and rarely exceed $\frac{1}{4}$–$\frac{1}{2}$ the size of the optic disc. In some of the cases, however, there are a few larger ones, but these are surrounded by a number of smaller patches or punctate spots. The spots vary much in shape, being usually nodular and irregular in outline, seldom stellate around the macula as in albuminuric retinitis. They are generally grouped between the disc and macula or near the macula, sometimes in the form of a semicircle or circle at some distance from it. De Wecker reports a case of this kind in his article on retinitis circinata (*Arch. d'Opht.*, January, 1894), which afterwards proved to be diabetic.

The color of the spots varies from a dull to a glistening, brilliant white or yellowish-white. The variation in the color

is undoubtedly due to the stage of the disease at which they are seen. These changes are accompanied by minute hemorrhages in most of the cases, which may be absorbed or give place later to foci of degeneration. Besides these changes in the central part of the retina, there may be small spots and hemorrhages to the nasal side of the disc or along the temporal vessels. The spots never run together, covering a large part of fundus, as they sometimes do in albuminuric retinitis.

In another class of cases very similar to these the whitish patches and hemorrhages are scattered over the whole fundus. The patches are larger in size (about that of O D) and are either uniform or interspersed with hemorrhages. De Wecker and Masselon give a good illustration of this class of cases in their *Text book of Ophthalmology*.

Œdema and active changes in the retina and optic nerve are rarely present although occurring so frequently in albuminuric retinitis. The vessels are usually of normal size and contour. This is one of the chief distinguishing features between diabetic and albuminuric retinitis.

Embolism of the retinal artery was present in two cases and thrombosis of the central vein in one case. These changes undoubtedly occurred in cases accompanied by endocarditis, which is so frequently present in the later stages of diabetes (Purdy). That there is a tendency to occlusion in the veins has been shown by the pathological examination made by J. Michel in which he found a partial occlusion of the central vein by a deposit on the inner wall.

The frequency of opacities and hemorrhages in the vitreous has not proved so great as would be supposed from literature, several authors mentioning it as a characteristic of the disease. It was present only seven times among the cases reported. In cases of central punctate retinitis they were very slight when present and the source could not be traced. In the hemorrhagic form they were more abundant and seriously affected vision. Their source could not be positively traced, and it was not known whether they came from the retina or from the vessels of the ciliary body.

The hemorrhagic form was less frequent than that with

foci of degeneration. Hirschberg has divided it into four kinds: (1) small punctate hemorrhages, (2) larger hemorrhages accompanied by opacities of the vitreous, (3) hemorrhagic infarction of the retina, (4) hemorrhagic glaucoma. We find the first kind to be most frequent and the least dangerous. The only cases reported as fully recovering were of this variety, but most of the cases later develop foci of degeneration in the spots where hemorrhages were and pass into the mixed form. It is rarely discovered in its early stages except by accident, as the disturbance of vision is very slight, sometimes being only an excessive sensitiveness to light, or flickering before the eyes. Seven cases of this kind were reported, one of which fully recovered. The second form is more serious, as the hemorrhages affect the vision, especially if they break into the vitreous. There is always danger of its developing into hemorrhagic glaucoma. Hemorrhagic infarction of the retina is rare, and the prognosis as to life of the patient is very serious. The only case occurring in pure diabetes was Case 14, and the patient died one year later. Only one case of hemorrhagic glaucoma was reported in pure diabetes, (Case 7), and this occurred after an iritis, but Hirschberg had three cases and Lagrange one, occurring when the diabetes was complicated by an albuminuria. The extensive vascular changes make these cases hopeless.

The *subjective symptoms* of this disease are very slight in the cases with minute hemorrhages and punctate spots in the centre of the retina. The retinitis may be accompanied or preceded by the diabetic amblyopia, which, according to Bouchardot is present in $\frac{1}{8}$ of all diabetic cases and frequently occurs without any fundus change. Most of the cases complain only of a dimness of the vision, especially for near work, and are seen only upon their applying for glasses to correct the defect. There may also be a sensitiveness of the eyes to light and a flickering before the eyes at an early stage of the trouble, due to an irritation of the retina. When the change affects the macula itself or there are large hemorrhages into the vitreous the visual disturbance is profound.

Of the *conditions following diabetic retinitis* the most frequent is atrophy of the optic nerve. Lagrange thinks that in almost every case the optic nerve is in a state of atrophy if it is not already completely sclerosed. It is quite different from that occurring with albuminuric retinitis which is preceded by a neuritis. The atrophy in diabetes is primary, due to the disturbance in nutrition or pressure from a hemorrhage into the sheath of the nerve.

The *pathology of diabetes itself* is not established, and we probably include several conditions, differing in their pathology and pathological anatomy. The changes which would affect the eye principally are those of the blood-vessels and nutrition. In the *Traité de Médecine*, 1893, vol. i., we find the following: "Endocarditis is present usually upon the auricular face of the mitral valve. The arteries of the brain and retina are frequently affected with fatty or atheromatous degeneration (Dupuytren, Charcot, Lecherché). Retinal hemorrhages are frequent, either due to the dyscrasia of the blood, to the fragility of the vessels, or to a simple atrophy, general or partial, of the retina and optic nerve."

Purdy (Diabetes 1889) states: "In most cases of diabetes there is an increased vascular tension as in chronic Bright's disease. It would seem that an extra demand is made upon the heart with primary hypertrophy of the left ventricle."

Osler (*Practice of Med.*, 1892) says: "The kidneys are sometimes fatty and show a hyaline change in the tubular epithelium particularly in the descending loop of Henle. This change also occurs in the capillaries. Endocarditis is very rare. The heart shows no characteristic changes."

Ludwig Mauthner, in an article upon the affections of the eyes in diabetes (*La France Médicale*, 1893), gives four means by which it affects the eye:

1. Subtraction of water from the tissue, with the presence of glucose (producing cataract, etc.).

2. Diminution of the resistance of the tissues, due to the general affection of the nutrition.

3. The presence of a toxic substance in the blood which produces the degeneration and inflammation.

4. Cachectic troubles. Affections of the nutrition would diminish the resistance of the blood-vessels so as to allow transudation of the blood corpuscles from the blood-vessels into the retinal tissues, but it is to the toxic substance in the blood acting upon the blood-vessels, especially the capillaries, to which most of the changes are due. These changes favor hemorrhages from the smaller vessels and the *localized* degenerations such as are possible with a system of terminal arteries without anastomoses. The optic atrophy, so frequently occurring with the retinitis, or alone, is due to the hemorrhages into the sheath of the nerve.

Of the *pathology of the ocular changes* only a few examinations have been made. The most extensive was the case (No. 8) reported by Stephen Mackenzie, the examination of the eye being made by Mr. Nettleship. It was briefly as follows:

There was a hyaline degeneration of the intima of the blood-vessels, especially the smaller ones, of the kidneys, spleen, liver, and some of the smaller vessels of the brain. This hyaline thickening of the walls was not evenly distributed but formed bulging masses extending into the lumen of the vessel. The epithelium of the secreting tubes in the kidney was granular and swollen. In the eye there was thickening of the retina in all its layers, but most in the nerve fibre layer where there were numerous varicose swellings. The disc was slightly swollen and the meshes formed by the separated nerve fibres were partly empty and partly filled by swellings similar to those in the retina. The swelling in other layers appeared to be the result of a chronic œdema with hypertrophy of the connective-tissue framework. The bacillary layer was wanting and its place was occupied in some parts by an albuminuric effusion. Here and there in the intergranular and molecular layers were round spaces (microscopical cysts) filled with faintly refracting globules (albuminous). The limitans interna was much thickened in parts and thrown into abrupt folds. No hemorrhages in retina. Vitreous in left eye contained a large quantity of blood which appeared to come from different spots in a gradual manner, but its source could

not be traced. The retinal arteries were greatly changed. Thickenings were present between the epithelium and the fenestrated membrane affecting only one side of the vessel and contracting the lumen very much. The changes affected all branches of the central artery but varied greatly in degree in different portions. As a rule the relative thickening of the coats was much greater in the very minute arteries than in those of larger size. At an early period the new material was more or less distinctly corpuscular and its inmost portion sometimes appeared composed of superimposed layers of endothelia. In many places where the disease was apparently of older date the appearance is best described as partly hyaline and partly fibrous, the hyaline changes being obvious in the smallest vessels. The muscular coat was not generally affected. The veins were merely distended, the coats not being altered. The capillaries of all parts were distended. In the retina were minute capillary aneurisms not very abundant but occurring in groups of about half a dozen. They were globular, apparently communicating with only one side of the vessel, never fusiform dilatations. They contained some reddish-brown granules and there were some granules just external to them suggesting that the hemorrhage into the vitreous may have been due to the rupture of a number of these minute sacs. The choroid was greatly thickened owing to distention of the vessels and infiltration.

In case No. 11, reported by Mr. Nettleship a microscopical examination of the eye was made by Mr. Lawford. There was thickening and puckering of the central artery, with the lumen almost obliterated. Veins thickened. Hyaline thickening of the vessels of choroid and some of the retinal vessels. Some large masses of waxy-looking granular material were observed in both granular layers, with smaller-sized ones in nerve-fibre layer. Capillary aneurisms. It was probably a true inflammation commencing around the vessels and accompanied by an exudate of inflammatory material into the retinal substance which pressed upon and destroyed the nervous tissue and finally underwent fatty degeneration.

In Michel's cases (No. 9) there was a double parenchymatous nephritis in stage of infiltration, hyperaemia of the brain, and a large caseous and ulcerating exudate in right lung. Microscopical examination of the eyes showed an infiltration of blood corpuscles between the nerve-fibre bundles and the separated nerve fibres of the right optic disc. There was partial closure of the lumen of the vena centralis for only a short distance ($\frac{1}{4}$ *mm*). The lumen was about $\frac{2}{3}$ filled by a mass firmly attached to the wall of the vessel. It consisted of a finely striated tissue, containing endothelial and lymph cells, with a few blood corpuscles.

Besides these, Galezowski examined case No. 6, where secondary glaucoma had developed. The sclera was bulged in numerous places, but there was no change in its structure. Fibrous exudate in choroid. Retina was detached and the eye filled with blood.

We see from these cases that the important changes are in the blood-vessels. These affect principally the small vessels, and are not so extensive as in albuminuric retinitis. The partial closure of the vessels tends to rupture, producing the small hemorrhages which are so common in this disease, and predominate over the degeneration which is the characteristic change in albuminuric retinitis. If there is anything in the pathology of the disease distinguishing it from other forms of retinitis it is the peculiar changes taking place in the small arteries and capillaries. In other forms of retinitis we have changes in the blood-vessels, but they are equally as great or predominate in the large vessels.

The *differential diagnosis* of this disease from others which resemble it is one of the principal objects of this paper. Of the two principal forms of the disease one is characterized by spots of degeneration alone or mixed with hemorrhagic spots, and the other by hemorrhages alone. The former is the most frequent and the most difficult to distinguish from albuminuric retinitis, which it closely resembles. Most authors say that it differs from it in some particulars, but give no definite points on which to differentiate. Lagrange, in his article, quotes the table prepared

by Badal for the differential diagnosis between the two diseases.

Diabetic Retinitis.	*Albuminuric Retinitis.*
1. Marked tendency to atrophy of the optic nerve.	1. Less tendency to atrophy, which occurs especially at a late stage.
2. Multiple hemorrhages, round and disseminated.	2. Hemorrhages equally multiple but elongated, and occupy especially the posterior layers.
3. Alterations diffuse.	3. Affect principally the circumpapillary and macular regions.
4. Apoplexies do not last to a late stage.	4. Last throughout life.
5. Small disseminated spots, with some exudate.	5. Whitish fatty spots, with infiltrations.
6. Color sense, nil.	6. Persists a long time.

Galezowski says that retinitis glycosurica presents, besides hemorrhages and fibrinous exudate, an alteration of the papilla, conducive, later, to atrophy. An optic neuritis is not found here as in albuminuric retinitis. Albuminuric and syphilitic retinitis are almost always binocular, while glycosuric retinitis is frequently monocular.

Noyes, as already quoted, says that the spots are more nodular and irregular than in the albuminuric form.

The principal differences which I should give as the distinguishing features between the diseases, as gathered from the cases quoted, I will briefly tabulate as follows:

Diabetic Retinitis.	*Albuminuric Retinitis.*
1. Groups of bright glancing spots in retina, irregular in outline, usually in central part, but frequently affecting whole of fundus.	1. At first a group of bright bluish-white spots in centre of retina, often forming stellate patch about the macula.
2. If the spots are larger there still exist small dots and lines, and they never run together.	2. Spots may run together and involve all of central part of retina.
3. Arteries and veins not much changed in appearance.	3. Arteries narrowed, veins large and irregular.
4. Optic nerve either not affected or atrophic.	4. Optic nerve swollen and outline indistinct.
5. Retina not diffusely affected.	5. Retina infiltrated.

The points of diagnosis are not well marked in all cases, and in some the appearance is that of the albuminuric form, with the tortuous vessels, swollen infiltrated nerve and

retina, and the grouping of the spots of degeneration about the macula. Under these circumstances nothing but an examination of the urine would allow one to conclude as to the cause.

The purely hemorrhagic form is less difficult to diagnose, for, with the small punctate hemorrhages, it presents a unique appearance, so different from the other forms of hemorrhagic retinitis, that it should suggest at least the presence of diabetes.

We have already seen that most cases of retinitis occur at an advanced stage of the diabetes, after other symptoms have called attention to the disease. In some of the cases, however, the notice of the patient was first called to their disease by the oculist, after examining the eyes. (See cases Nos. 29 and 46.)

As to the *prognosis* regarding the life of the patient, the occurrence of the retinal changes has been considered a grave symptom. Yet in most of the cases where the histories were followed the patients were alive several years after, and in some the diabetes was entirely cured.

As to the *treatment* of the disease there is little indication to do anything else than combat the constitutional dyscrasia by proper diet. In two of my cases the ocular symptoms appeared a few months after discarding the antidiabetic diet prescribed and returning to their old ways of living. In one of them the vision improved upon returning to a strict diet without the starchy foods. Leber recommended the administration of carbolic acid, but other writers have found that while it may have a beneficial effect in diabetic amblyopia, it does not influence the course of the retinitis.

Galezowski considers the disease curable. "At the beginning an antiphlogistic treatment (bleeding behind the ear, etc.) should be used. After the congestive stage terminates, resolving remedies (such as eserine, injections of pilocarpine, collyria of iodide of soda and potassium 1 per cent.) should be combined with the general antidiabetic treatment.

LIST OF CASES.

Case No.	Sex.	Age.	Duration of Diab.	Dur. of Eye Trouble.	Condition of Fundus.	Reference.
1	F	46	3 yrs.		Opacities in vitreous. Large and small bright spots scattered over whole of fundus, mixed with retinal hemorrhages partly old and partly fresh.	Desmarres, *Traité des mal. des yeux.* 1858.
2	M	55	5 yrs.	4 mo.	Several large white patches (½ to 2 times O D) surrounded by a number of smaller white patches and hemorrhages.	Desmarres, *Traité des mal. des yeux*, 1858.
3	F	60	3 yrs.		Had iritis both eyes a few months ago. Whitish glistening spots in the retina between the O D and macula forming circle around macula. They look like fatty degeneration and similar to albuminuric retinitis. A few hemorrhages. Slight haziness of vitreous.	H. D. Noyes, *Trans. Am. Ophth. Soc.* 1869.
4	F	29	5 yrs.	14 days	Diffuse opacity of vitreous. Papilla grayish white and not clearly demarcated. Retina hazy in central part. Arteries thin, veins large. A large number of small round and irregular hemorrhages. No whitish spots, but some appeared later in places where hemorrhages were.	Haltenhoff, *Zehender's klin. Mon.* 1873.
5					Upper nasal branch of central artery is obliterated and degenerated to a whitish string. Numerous extravasations of blood and some whitish spots.	Galezowski, *Recueil d'Ophtal.* 1873.
6					Hemorrhages and whitish extravasations in retina. Later iritis serosa and acute glaucoma. Microscopical examination made. (See page 216).	Galezowski *Receuil d'Ophtal.* 1873.
7	F		2 yrs.		Fundus is opaque showing œdema of the retina or an exudate between memb. limitans interna and hyaloid membrane. There are whitish spots of exudate and striped hemorrhagic extravasations along the arteries in macular region. Hemorrhages were absorbed and exudate disappeared.	*Umé Arch. méd. Belges* 1875.
8	F	52	1 yr.	3 mo.	Has two large floating films in right eye from blood clot. Vitreous turbid. An opaque linear projection into the vitreous was seen, on which retinal vessels were visible and to which one of the filmy floating webs is attached. Left eye vitreous has the appearance of a diffuse hemorrhage. Only a glistening white crescentic spot was seen near disc but not in connection with it. Died three months later. P M examination made. (See page 214).	Stephen Mackenzie, *Ophth. Hosp. Rep.*, vol. ix., 1877.
9	M	21	1 yr.		Right eye showed a large round hemorrhage upon O D. Much emaciated. Died soon after examination. P M examination made. (See page 216).	Michel, *Deutsche Arch. f. klin. Med.* 1878.
10	F	6		4 wks.	Small white spots at outer side of O D near macula similar to albuminuric retinitis. Outlines of disc obscure. Arteries and veins slightly smaller than normal and papillæ red on outer side. Media clear. No hemorrhages. Under strict diet and treatment recovered. Right eye ended in atrophy of retina and optic nerve. Diabetes was secondary to malaria.	Culbertson, *Detroit Lancet*, 1882.
11	F	63	18 yrs.	Rt. eye 2 yrs. Lft. eye 1 day	Right cholesterine crystals in vitreous. Large number of yellowish white spots of different sizes in retina in region of macula. No haze of retina or hemorrhages. Spots look old. L E yellowish patches not so abundant as in right. Diffuse haze of central part of retina and O D as in embolism of retinal artery. Main lower branch of artery is obliterated by plug at its origin, near O D. Numerous small hemorrhages up to periphery. Has slight trace of albumin in urine. Died six months later from gangrene. P M examination made. (See page 215).	Nettleship, *Trans. of Ophth. Soc. of the U. K.* 1882.

Case No.	Sex.	Age.	Duration of Diab.	Dur. of Eye Trouble.	Condition of Fundus.	Reference.
12	M	50		1 yr.	Has large number of spots and patches of whitish degeneration in central part of retina, similar to albuminuria retintitis but not grouped the same. They are more scattered and not spoke-shaped. Spots lie beneath blood-vessels never above them. They are somewhat yellower than albuminuric retinitis.	Nettleship, *Trans. of Ophth. Soc. of the U. K.* 1886.
13	M	56	Health good		L E has small grayish spots in region of macula. R E has hemorrhages near O D.	Hirschberg, *Deutsche med. Woch.* 1887.
14	F	71			Has hemorrhagic infarction and complete plugging of central vein. Numerous hemorrhages over whole of fundus. Hemorrhages absorbed later leaving spots of degeneration. Died one year later.	Hirschberg, *Deutsche med. Woch.* 1887.
15	M	64	Sev.yrs.		Cat. right eye. Left eye has central retino-choroiditis around macula and presents all the appearance of alb. retinitis.	Lagrange, *Archiv. d'opht.* 1887.
16	F	65	17 yrs.	2 yrs.	Slight infiltration of papilla. Small spots of exudation around papilla with reflection like mother-of-pearl interspersed with small hemorrhages. In several places large plaques of choroidal atrophy. Floating opacities in vitreous.	Lagrange, *Archiv. d'opht.* 1887.
17	F	53	5 yrs.		Right eye œdema of papilla with small hemorrhages around it. Left eye papilla excavated and in state of atrophy without increase of tension.	Lagrange, *Archiv. d'opht.* 1887.
18	M	53	3 yrs.		Small ecchymoses around the macula which disappeared in a few weeks. Central scotoma L E. Vision became normal.	Lagrange, *Archiv. d'opht.* 1887.
19	M	40		1 mo.	Hemorrhagic retinitis characterized by small hemorrhages scattered around papilla. Papilla swollen and irregular in outline. In places the small vessels are covered by the spots in question.	Lagrange, *Archiv. d'opht.* 1887.
20	M	67	3 yrs.		Small hemorrhagic spots in region of macula and upon O D. Until death four years later alternate amelioration and aggravation of the trouble.	Lagrange, *Archiv. d'opht.* 1887.
21	F	62	Sev.yrs.		Left eye nearly blind from numerous hemorrhages in the vitreous. R E central corona of white (mother-of-pearl) spots around macula similar to albuminuric retinitis. Large number of small hemorrhages in posterior part of fundus.	Lagrange, *Archiv. d'opht.* 1887.
22	F	57		2 yrs.	Central retinitis both eyes which has all the appearance of albuminuric retinitis. Exudative corona around papilla interspersed with small hemorrhages. Beginning posterior polar cataract both eyes.	Lagrange, *Archiv. d'opht.* 1887.
23	F	63	Sev.yrs,		Central retinitis with spots and hemorrhages around macula like albuminuric retinitis.	Lagrange, *Archiv. d'opht.* 1887.
24	M	53	7 yrs.		Hemorrhages radiating from papilla both eyes. Whitish spots in region of macula.	Lagrange, *Archiv. d'opht.* 1887.
25	M	51	5 yrs.	Sev.mo.	Left eye vision entirely obliterated. Small hemorrhagic spots irregularly distributed around macula. Optic papilla in stage of atrophy. Floating opacities in vitreous. R E exudative retino-choroiditis in region of macula, small spots of hemorrhage, commencing atrophy of O D.	Lagrange, *Archiv. d'opht.* 1887.

Case No.	Sex.	Age.	Duration of Diab.	Dur. of Eye Trouble.	Condition of Fundus.	Reference.
26	M	48	4 yrs.	2 yrs.	Left eye became blind one year ago from secondary glaucoma after plastic iritis with occluded pupil. Several enlarged blood-vessels in iris. Right eye has retinitis with haziness. Near macula are yellowish, densely opaque spots such as described in Case 12. Retinal veins distended and scattered hemorrhages. Near fovea centralis 2-3 vessels of medium size appear in retina as though they came from choroid. They form a fine plexus extending into the vitreous.	Nettleship, *Trans. of Ophth. Soc. of the U. K.* 1888.
27	M	49	3 yrs.		Hemorrhages and white patches in retina scattered over whole of fundus. Vitreous clear.	James Anderson *Ophth. Rev.* 1889.
28	M	70	1½ yrs.		Opacity of lens. Two large white spots between O D and macula and remains of old hemorrhage.	James Anderson *Ophth. Rev.* 1889.
29	M	45			Health good. Complains of weakness of accommodation. There was one small hemorrhage in central part of left fundus. Two years later there were small indistinct spots and hemorrhages over whole of fundus. Hemorrhages are partly round and partly elongated.	Hirschberg, *Deutsche med. Woch.*, 1890.
30	M	53	6 yrs.	8 days	Complains of recurring attacks supposed to be glaucoma. Has bright spots of degeneration above and at some distance from papilla, also round and pointed hemorrhages.	Hirschberg, *Deutsche med. Woch.*, 1889.
31	F	58	3 yrs.	4 wks.	Large number of bright spots in central part of retina and a few hemorrhages. A single bluish white spot of exudate.	Hirschberg, *Deutsche med. Woch.*, 1890.
32	M	65		2 mos.	There were a dozen small white spots between O D and macula. A small group of such spots a short distance from O D and a single small hemorrhage in the centre of retina.	Hirschberg, *Deutsche med. Woch.*, 1890.
33	M	53		3 mos.	Macular side of O D is pale. Delicate white spots (round and striped) like fatty degeneration in centre of retina both eyes.	Hirschberg, *Deutsche med. Woch.*, 1890.
34	M	65	3 yrs.	4 wks.	Right eye has delicate changes. A few small hemorrhages with occasional white spots. L E extensive central opacity of retina with larger bright spots and hemorrhages along the enlarged veins.	Hirschberg, *Deutsche med. Woch.*, 1890.
35	M	63	6 yrs.	1 yr.	Lenticular opacities. Retina has delicate fine opacities along some of the vessels; to the side of this small bright points, small hemorrhages, and one large brown spot from former hemorrhage, in centre of fundus.	Hirschberg, *Deutsche med. Woch.*, 1880.
36	M	68			After cataract extraction examination showed clear media, small hemorrhages, and white spots in centre of retina and around O D.	Hirschberg, *Deutsche med. Woch.*, 1890.
37	M	63		2 yrs.	Became near-sighted two years ago. Lens opaque. Small hemorrhages and glistening white spots in central part of retina.	Hirschberg, *Deutsche med. Woch.*, 1890.
38	F	69	3 yrs.		Opacity of lens. Small whitish spots and small hemorrhages here and there in central part of retina.	Hirschberg, *Deutsche med. Woch.*, 1890.
39	M	56	Long time.		L E has group of fine spots in central part of retina near O D. R E has a hemorrhage on O D.	Hirschberg, *Deutsche med. Woch.*, 1890.
40	F	62	8 yrs.		Lens opaque. Whitish opacities in centre of fundus similar to senile retinitis.	Hirschberg, *Deutsche med. Woch.*, 1890.

Case No.	Sex.	Age.	Duration of Diab.	Dur. of Eye trouble.	Condition of Fundus.	Reference.
41	F	48	3 yrs.		Small punctate hemorrhages in retina. L E has had iritis.	Hirschberg, *Deutsche med. Woch.*, 1890.
42	M	43	6 mo.		Four bright sharply defined round spots about 2 to 25 *mm* in size and a few small white ones with hemorrhages were situated outward from macula. Later other hemorrhages appeared and a vessel which ran underneath had a mottled appearance.	Seggel, *Münch. med. Woch.*, 1891.
43	F	64	Sev. yrs.		Peripheral striæ in lenses. Typical diabetic retinitis. Intensely bright white spots arranged around macula in streaks and lines, perhaps approaching the stellate appearance of alb. retinitis more than usual. Other spots of degeneration less marked and finer farther from macula though few extend beyond O D. Hemorrhagic spots scattered among white ones to nasal side of papilla. Papilla and vessels normal.	Fridenwald, *Arch. of Ophth.*, 1891.
44	M	64			Cataract of one eye. The other eye had numerous small blood spots in retina over nasal part of fundus. Vitreous clear. V = $\frac{18}{18}$. Urine 4 per cent. sugar, no albumin. Three months later the fundus was normal; no hemorrhagic spots. One month later a few fresh hemorrhages, and yet five months later a large hemorrhagic spot appeared near papilla. Died two years after first examination.	F. C. Hotz, Report of case kindly furnished me.
45	M	55	5 yrs.	6 mo.	Right eye has medium-sized white patches scattered over whole of fundus. In the left a number of whitish spots are interpersed with hemorrhages. The changes are more recent than in right. Vitreous clear.	Case seen by Author in clinic at Moorfields, London.
46	F	52	4 yrs.	Recent	Has diffuse opacity of vitreous both eyes. In right eye there was a small whitish spot of exudate between the optic disc and macula and one in upper part of fundus with a few small round hemorrhages. Left eye had only one small spot of exudate near macula. Veins large and fundus appeared hyperæmic. V = $\frac{20}{18}$ each eye.	Case seen in Author's practice, Dec. '93.
47	F	62	6 yrs.	6 mo.	R E has a number of brilliant white spots in macular region, one large one ($\frac{1}{4}$ size *od.*) surrounded by a number of small punctate spots. A few hemorrhages along the vessels in the outer part of fundus. L E has a number of small white spots scattered around macular region. Two weeks later the hemorrhages in right eye had disappeared, their place being occupied by whitish spots of degeneration. Three months later the vision was considerably improved but the fundus conditions were similar. Patient has some albumin in urine.	Case referred to Author by Dr. Wilder, Ill. Eye and Ear Inf.

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ALVEOLAR FIBRO-SARCOMA OF THE OPTIC NERVE.[1]

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(*With five figures in the text.*)

I AM indebted to Dr. E. Lopez, of this city, for the pathological specimen which constitutes the subject of this paper. He has furnished me with the following

CLINICAL HISTORY.

On June 25, 1890, Dr. Lopez was consulted at his office by a lady, fifty-five years of age, for a protrusion of the right eyeball this had commenced about a year before, and had since then increased continuously; there was no pain or inflammatory reaction connected with this and it could not be assigned to any particular cause. *St. Praes:* The right eyeball protruded in a directly forward direction, being almost out of the orbit; it was immovable; the pupil was directed upwards and outwards; it was dilated and did not react to light; the lens showed an incipient opacity; there were also floating opacities in the vitreous. The ophthalmoscope revealed an optic neuritis, the margins of the disc were obliterated, the retinal vessels being only visible at some distance from the disc. T>n. H + 4 D (with ophthalmoscope) V = o. Left eye: Tn. Myopia − 3 D. V = $\frac{1}{3}$ (with correction). Only noticeable lesion was an incipient opacity of the lens. *Diagnosis:* Tumor of the optic nerve or its sheaths (RE).

The general health of the patient being good, and in view of her pusillanimity, Dr. Lopez recommended a purely palliative treatment till the progress of the disease should make the patient understand the necessity for operative interference.

[1] Studied in the laboratory of Dr. C. M. Desvernine.

The patient was again seen by Dr. Lopez six months later; there were now severe neuralgic pains in the whole area supplied by the ophthalmic branch of the fifth nerve; the eyeball was entirely out of the orbit, the lids closing behind it; the conjunctiva was congested, swollen, œdematous; the cornea had sloughed; the projection forward of the eyeball amounted to *2 cm.*

The next day, aided by Drs. Larios and Silverio, Dr. Lopez, after performing a preliminary enucleation, extirpated a hard cylindrical tumor, which occupied the centre of the orbit. The tumor had produced a dilatation of the optic nerve, which had attained a diameter of 1 *cm*; it extended from the eyeball to the optic foramen.

The patient recovered from the operation, but a year later died with cerebral symptoms; for some time previously she had been idiotic; the left eye had also been blind for some time, showing a total atrophy of the optic nerve.

HISTOLOGICAL EXAMINATION.

I did not receive the pathological specimen till 1891, and through unavoidable circumstances was unable to study it till 1894. The tumor had been cut up into a number of small semi-cylindrical pieces, it being impossible to determine the topographical relation in which they stood to one another; the eyeball, which had lain all this time in alcohol, was shrivelled up.

On laying the eyeball open by an equatorial section I found that the tumor had extended into the interior of the eye; at the papilla it formed a prominent mushroom-shaped projection, which was raised about 5 *cm* above the surface of the adjacent retina; its passage into the latter was rather abrupt (Fig. 1). The retro-

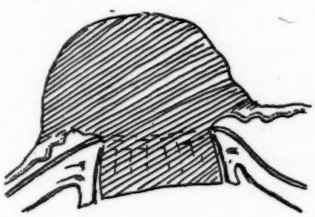

FIG. 1.

Section through optic-nerve entrance, showing the projection of the tumor into the interior of the eye.

bulbar tumor was cylindrical, softish, granular, and was surrounded by a thick fibrous capsule; its diameter was about 1 *cm.*

MICROSCOPICAL EXAMINATION.

(A) *Retrobulbar Tumor.* This does not present the same structure at all points. In a few sections it is more highly organized. From the thickened and dense dural sheath numerous connective-tissue septa extend into the interior of the tumor, which dividing and uniting with the subdivisions of other septa divide the mass of the neoplasm into a large number of alveoli (Fig.

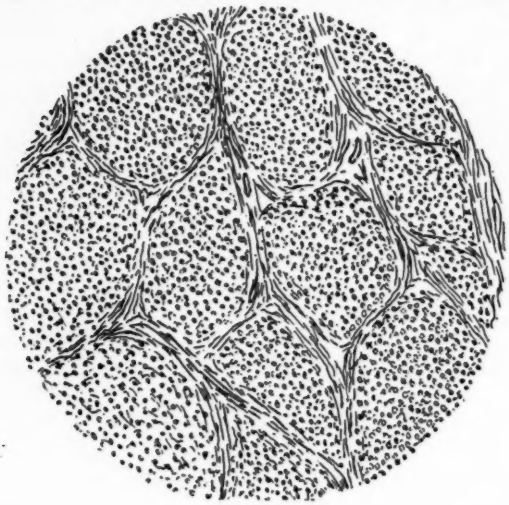

FIG. 2.
Portion of retrobulbar tumor showing alveolar structure.

2), which are filled with closely packed cells; these are mostly spindle-shaped, some, however, are round and others polyhedral. The spindle cells, which constitute the bulk of the tumor, have not everywhere the same appearance; in *some few* of the alveoli they contain more protoplasm and are somewhat wider than is usually the case in an ordinary spindle-celled sarcoma; between these there is a very small amount of intercellular substance. These cells remind one somewhat of endothelial cells, although their appearance is by no means characteristic. The great majority of the alveoli are filled with ordinary sarcoma spindle cells of different size with a moderate admixture of round cells and leucocytes, and between them (within the alveoli) there is a delicate but distinct fibrillar intercellular substance. The fibrous

septa contain a large number of blood-vessels and are rich in cells. There is everywhere an intimate connection between the cells within the alveoli and those constituting the stroma.

In the vast majority of the sections this alveolar structure is absent, the tumor consisting of a dense accumulation of smaller and larger fusiform cells, intermingled with a relatively large proportion of round cells and moderately infiltrated by ordinary white blood corpuscles (Fig. 3). The arrangement of these cells is ex-

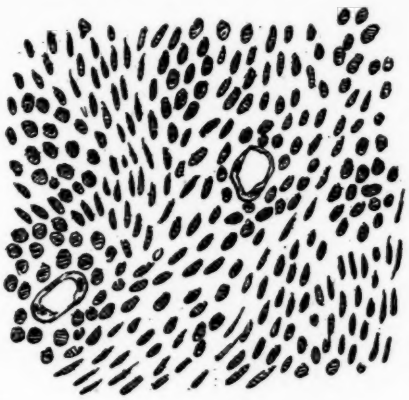

FIG. 3.

Portion of retrobulbar tumor, showing more general atypical fibrosarcomatous structure.

tremely irregular—at places they are grouped together in bands which cross each other at all possible angles—at places the arrangement follows no particular rule—in the peripheric portions the cells affect a more or less concentric arrangement. There is a delicate intercellular fibrous network which at places is very prominent, reminding us of a young fibroma, and which at places is completely masked by the cells; between these extremes we find the proportion between cells and intercellular substance varying greatly. Blood-vessels are to be found scattered throughout the tumor; their walls are thick. At places the spindle cells are arranged more or less concentrically around them (as seen when the vessels appear cut perpendicularly to their axis) or along the vessel walls, the long axes of the spindles running parallel with that of the vessel (when the vessels appear in longitudinal section), forming sheaths for the vessels; their endothelium is everywhere unaltered.

The fibrous capsule of the tumor is formed by the thickened dural sheath, and is made up of dense fibrous tissue. In only one slide is an extension of the neoplasm into the neighboring orbital cellular tissue noticeable—that was at a point of exit of a blood-vessel, the thickened walls of which as well as the immediately surrounding cellular tissue are infiltrated with sarcoma spindles; this infiltration is limited to the cellular tissue just outside the sheath; the lumen of the vessel was quite free.[1] Nowhere in this portion of the tumor can any trace of nerve-fibre be found.

In some places the intervaginal space is free, in others no space can be discovered between the thickened dural sheath and the mass of the neoplasm.

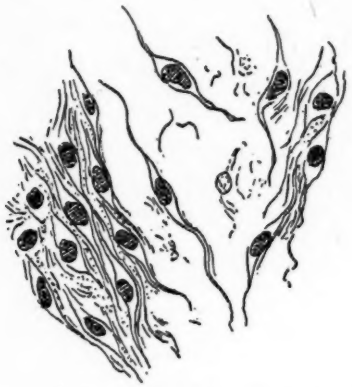

FIG. 4.
Highly magnified section through intraocular tumor.

(*B*) *Intraocular Tumor.*—This portion of the tumor presents a totally different aspect; the cells are no longer as closely packed together, but wider apart, and between them the cellular interspaces are occupied by granular matter and by an *abundant fibrillar network proceeding from the cell processes.* The cells are mostly spindle-shaped, of which there appear to be two varieties: (*a*) one smaller and wider, (*b*) one longer and more slender; they are bipolar, with long fibrillar processes, which mingling with those of the neighboring cells form the fibrillar meshwork just mentioned (Fig. 4). There is also an abundant admixture of round cells and

[1] This bears out Dr. Holden's views on the extension and development of sarcoma by a proliferation of the adventitial cells of the vessel walls (ARCH. OF OPHTH., xxii., 3, 289).

of leucocytes. This portion of the tumor occupies almost exclusively the superficial layers of the retina, especially the nerve-fibre layer, from this portion extending secondarily to the deeper-seated; these are quite recognizable and only show some ordinary round-cell infiltration, and here and there some extension of the tumor cells between their normal elements (Fig. 5). This intraocular

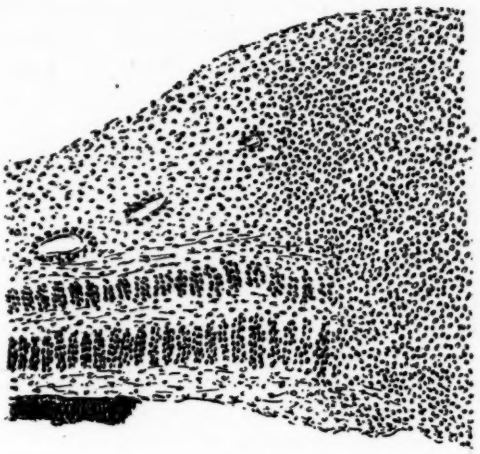

FIG. 5.
Section showing invasion of retina by neoplasm.

extension of the tumor is moderately vascular, the blood-vessels showing thickened walls with fusiform cells accumulated around them. At one or two places near the optic-nerve entrance the retina is raised from the choroid by extravasated blood. The extension of the neoplasm into the interior of the eye via the interstitial tissue of the optic nerve is very apparent. At the optic-nerve entrance the intervaginal space is quite free.

The choroid is normal, at least as far as relates to an involvement by the neoplasm.

HISTOLOGICAL CONSIDERATIONS.

It is curious to note the differences in structure presented by this tumor at different points. The great bulk of the neoplasm is evidently a fibro-sarcoma of the small spindle-cell type, in which the structure is as usual atypical; but then we have a portion presenting a curious alveolar structure,

the cells within some of these alveoli reminding one in their appearance of endothelial cells. This appearance is as already stated not sufficiently characteristic to warrant a definite conclusion as to their nature. Should they be considered as endothelial cells, the tumor would come under the "endothelial sarcomata" of Birsch-Hirschfeld[1]; but this is a very delicate point to settle, and I will classify the growth as an "alveolar fibro-sarcoma." It is a pity that one cannot determine the relative position of these two portions of the tumor. The still greater alteration in structure presented by the neoplasm on its extension into the interior of the eye, is readily explainable by the different physical and biological conditions under which the cells find themselves (resistance, nutriment, soil, etc.). The spreading apart of the cells, the fibrillar meshwork proceeding from the cell processes and granular substance contained in the cellular interspaces, lead me to consider this portion of the new growth, especially that bordering on the vitreous as undergoing *myxomatous degeneration*; no mucin can be found in the cellular interspaces, but this may possibly be explained by the long sojourn in alcohol. This transition from the more highly organized alveolar sarcoma to the more atypical ordinary variety, and into the still more lowly organized myxosarcoma, is very instructive to follow.

There is another example of this kind reported by Neumann,[2] in which an optic-nerve tumor presented a partly alveolar and partly compact structure, in which there were

[1] This author describes a variety of tumor often erroneously described as "endothelioma," which occur most frequently in the *membranes of the brain*, and which should be properly considered as sarcoma. They present, in a fibrillar (or myxomatous), more or less cellular stroma, accumulations of endothelial cells. He also mentions the possible occurrence of bands of endothelial cells in sarcomatous tumors of different organs, which proceed apparently from a proliferation of the connective-tissue endothelium or of that of its lymphatics. He considers that these tumors should be denominated "endothelial sarcomata," in view of their preponderating cell growth, these tumors clinically fitting into the "fibro-sarcomata." Birsch-Hirschfeld also seemingly differentiates between the "endothelial alveolar sarcoma" and the endothelioma proper (endothelial cancer).—(*Lehrbuch der alg. path. Anatomie*, Birsch-Hirschfeld, Leipsic, 1889, p. 185).

[2] *Arch. f. Heilk.*, xiii., 310.

concentrically arranged collections of fusiform cells. The compact portion consisted of thick fibrils containing sarcoma cells in their meshes; the centre of many of the cells was encrusted; the fusiform cells were endothelial. Neumann calls his tumor a psammoma on account of the cellular encrustations.

GENERAL CONSIDERATIONS.

On looking up the literature of optic-nerve tumors, I find that I have been able to gather together a much larger number of cases than previous compilers. Willemer[1] collected 27 cases, Vossius[2] 34, Jocqs[3] 62, Braunschweig[4] 61, whilst I have managed to collect, either directly or through extracts, reports of 117 (including the one here described). I have arranged these cases in a tabular form at the end of this paper, noting the points of most interest as regards nature, age, duration, operative interference, recurrence, etc. Among these I have included 5 cases which rightly do not belong to the optic-nerve tumors,—*i.e.*, 3 cases of large calcareous concretions in the optic nerve, and 2 cases of aneurism of the central artery of the retina; on the other hand, I have made no mention of Jacobson's case,[5] as here there was no optic-nerve tumor proper. These tumors may be grouped together as follows:

Calcareous concretions	3	*Endothelial sarcoma*	1
Aneurisms of the cent. art. of the ret.	2	Endotheliomata	5
Cysts[6]	2	Gliomata	3
Fibromata	9	Myxo-glioma	1
Myxomata	13	Glio-sarcoma	2
Myxo-fibromata	3	Carcinomata (metastatic)	3
Sarcomata[7]	12	Gumma	1
Fibro-sarcomata	4	Tubercular tumors	4
Myxo-sarcomata	14	Tumors with no or doubtful denomination	33
Alveolar sarcomata	2		53
	64		
Total	117		

[1] *Arch. f. Ophth.*, xxv., 1, 16.
[2] *Arch. f. Ophth.*, xxviii., 3, 33, 283.
[3] *Thèse de Paris*, 1887; remarkably complete work.
[4] *Arch. f. Ophth.*, xxxix., 1.
[5] *Arch. f. Ophth.*, x., 2, 95.
[6] One was a hydatid.
[7] Among these is included Dusausay's angiolithic sarcoma (*Bull. de la Soc. Anat.*, xx., 1875), which possibly would be better included among the endothelial sarcomata.

In these, as in other statistics, one can readily see the immense preponderance of tumors of the connective-tissue type, which here seem to be especially prone to myxomatous degeneration. As regards the special variety of sarcoma to which our case belongs, we find 2 other cases—one of Chenantais,[1] described as a medullary alveolar neuroma, and Neumann's (already mentioned)—which, I think, may safely be included in Birsch-Hirschfeld's "endothelial sarcomata." (Endotheliomata are the tumors which are most likely to present an alveolar structure; thus it is to be found in 3 of the 5 cases reported—in Knapp's,[2] in Alt's,[3] and in Evetsky's.[4])

The extension of the tumor into the interior of the eye is a very rare occurrence, we only find it recorded in four cases: (1) in a case of Goldzieher's [5] of glioma of the optic nerve and retina, in which the optic-nerve tumor may very well have been secondary to the retinal; (2) a case of Knapp's [6] of sarcoma of the orbit, piercing the optic-nerve sheath and extending along the optic nerve into the eye, where it spreads in the nerve-fibre layer of the retina; (3) a case of Armaignac [7] of encephaloid of the retina and optic nerve (glioma), in which the retinal growth was most probably the primary one; and (4) a case of Sattler's [8] of a tubercular tumor of the optic nerve which extended into the eye along the nerve. Jacobson's case, usually quoted as a case of extension of an optic nerve-tumor into the interior of the eye, does not, as already stated, belong here, as in it there were scattered myxomata in the orbit with metastatic foci in the eye, and no optic-nerve tumor proper.

Clinical Features of the Case.—There is nothing of special interest about these. The symptoms present: exophthalmos in a directly forward direction, early loss of sight, painless course, optic neuritis, hypermetropia, increased tension,

[1] *Soc. Anat. de Nantes*, 1879.
[2] ARCH. OF OPHTH. AND OTOL., iv., 323. This case was described as a carcinoma, but a careful study of the case and drawings leads me to conclude that in all probability it was an endothelioma. At the time when the case was reported endothelioma had not yet been properly studied.
[3] *Arch. f. Augenheilk.*, vii., 1, 46.
[4] *Arch. f. Augenheilk.*, xii., 1, 16.
[5] *Arch. f. Ophth.*. xix., 3, 134.
[6] ARCH. OF OPHTH. AND OTOL., v., 132.
[7] *Journ. de méd. de Bordeaux*, 1878
[8] *Arch. f. Ophth.*, xxiv, 3, 127.

etc., being common to most optic-nerve tumors; generally there is more mobility preserved than in this case. It would have been interesting to note the ophthalmoscopic picture presented by the nerve head on its invasion by the neoplasm. It is a pity that during the later stages of the disease the opacity of the cornea prevented an ophthalmoscopic examination; an addition to Jacobson, Knapp, and Sattler's descriptions would have been instructive.

STATISTICAL CONSIDERATIONS.

I think it may be of interest to some if, before terminating, I call attention to a few points of general statistical import as regards the age of appearance and duration of these tumors, as well as to the results obtained by the different methods of operative interference as inferred from the appended tabular report.

Age.—Optic-nerve tumors have been found at all ages; from almost immediately after birth to sixty-seven years of age. In the sixty-seven cases where the age is mentioned we find:

Before 10 yrs.	30 cases.
Between 10 and 20 yrs.	16 "
" 20 " 40 "	14 "
" 40 " 60 "	5 "
After 60 yrs.	2 "

Youth is thus seen to be a marked predisposing factor; this is especially true for tumors of the sarcoma type. All the gliomata except one occurred in patients under 10; the one exception was a glio-sarcoma.

Duration.—The duration of the tumors before operative interference or before death, is mentioned in 50 cases. It fluctuates between 2 months and 20 years,—the average duration being 4.61 years. The course is apt to be slower with myxomata, endotheliomata, and fibromata. The following table will show the relative duration of each kind of tumor:

NATURE.	EXTREME DURATION.	AVERAGE.
Fibromata [1]	From 4 mos. to 20 yrs.	5.20 yrs.
Myxomata	" 2 yrs. to 14 "	7.10 "
Sarcomata [2]	" 3 mos. to 18 "	3.47 "
Gliomata	" 6 " to 2 "	1.50 "
Endotheliomata	" 3 " to 8 "	5.83 "
Carcinoma	" to 8 mos.	.66 yr.
Tubercle	" 2 " to 6 yrs.	3.08 yrs.
Tumors without denomination	" 2 yrs. to 8 "	4.40 "

[1] We include in this group the myxo-fibromata.
[2] We include in this group the fibro-, myxo-, and endothelial sarcomata.

Operative Results.—Three principal operative procedures have been employed in dealing with these tumors; (*a*) exenteration of the orbit; (*b*) extirpation of the eyeball and tumor; (*c*) extirpation of the tumor with preservation of the eyeball. An operation is recorded in 68 cases. An exenteration was performed in 3 cases, all of which recovered, and in none of which there was any recurrence.

In 54 cases both eyeball and tumor were extirpated; mention of the subsequent course is made in only 25 cases; there was no recurrence in 10, the neoplasm recurred in 8 cases, and in 7 the operation was followed by death within a few days, generally with cerebral symptoms. This can in most cases be attributed to sepsis. In 4 of these cases metastatic growths or an extension into the brain were, however, found. It is safe to infer that there was no recurrence in the majority of the 29 cases where no mention is made of the subsequent course.

The eyeball was preserved in 11 cases.[1] Knapp is the surgeon who has performed most of these. There was no recurrence in 4 cases; one case was followed by death within a few days. The eyeball was subsequently lost or had to be enucleated in 4 cases, in one of which the tumor recurred, and in one of which the operation was followed within a few days by death. In this case there was an intracranial extension of the growth.

We find mention made of operations performed on recurring tumors in 4 cases: 2 recurred again, and in 2 the operation was followed by death within a few days. In one of these an intracranial extension was found at the autopsy.

Thus we find in 68 cases 11 relapses and 11 deaths. The relapses may, in view of the poor statistics yielded by the secondary operations, be considered to have terminated fatally. The recurrences occurred in the cases of: 1 fibroma[2]

[1] Critchett, *Med. Times and Gazette*, 1832; Knapp, ARCH. OF OPHTH. AND OTOL., iv., 323; Knapp, *Ophth. Soc.*, Heidelberg, 1874; Knapp, ARCH. OF OPHTH. AND OTOL., v., 132; Grüning, ARCH. OF OPHTH. AND OTOL., v., 508; Schott, *Arch. f. Augenheilk.*, vii., 1–81; Alt, *Arch. f. Augenheilk.*, vii., 1, 46; Strawbridge, *Trans. Am. Ophth. Soc.*, 1878; Knapp, *Trans. Am. Ophth. Soc.*, 1879; Rohmer, *Soc. franç. d'opht.*, Mai 2–5, 1892, *Ann. d'Oc.*, cvii., 354; Lagrange, *Congrès franç. d'opht.*, April, 1892, *Ann. d'Oc.*, cvii., 377.

[2] A secondary operation performed on the recurring growth was followed within a few days by death.

(of 20 years' duration); 1 sarcoma; 1 fibroma-sarcoma (of 2 years' duration); 1 myxo-sarcoma; 1 endothelioma (of 8 years' duration); in 1 case of tubercular tumor,[1] 1 glioma,[2] 2 of unnamed tumors (one of 4 years' duration). The deaths occurred in: 2 sarcomata (one of 3½ years' duration); 1 fibro-sarcoma (of 2 years' duration); 2 myxo-sarcomata (of 3 and 5 years' duration); 1 endothelial sarcoma (of 18 years' duration); 2 gliomata (one of 2 years' duration); 1 glio-sarcoma (of 2 years' duration), and the 2 cases following secondary removals.

These statistics may be summarized as follows:

Operation.	Recurrence.	Death from Operation.	Recorded Non-Recurrence.
Exenteration of the orbit, 3 cases.			3 cases.
Extirpation of tumor and eyeball, 54 cases.	8 cases.	7 cases.	10 "
Extirpation of tumor, with preservation of eyeball, 11 cases.		1 case.	4 "
Subsequent loss of eyeball, 4 cases.	1 case.	1 "	
Secondary operation, 4 cases.	2 cases.	2 cases.	

CONCLUSIONS.

These statistics teach us that an operative interference is called for as soon as the diagnosis is made, as the tumor, if left to itself, will almost necessarily produce death, and the earlier the operation is performed the greater the chance of success. The comparatively large proportion of deaths immediately after the operation makes a strict adherence to the rules of aseptic and antiseptic surgery a necessity. The presence of metastatic growths within the cranium or of a direct intracranial extension seems to predispose to infection of the meninges; if such an extension can be inferred, all operative interference, with a radical cure in view, is contra-indicated.

[1] This should not be considered as a real recurrence. The patient died after some time of a tubercular meningitis.

[2] In this case three operations were successively performed, the three being followed by a recurrence of the growth.

In view of the good statistical returns, an attempt at preserving the eyeball is justifiable and even recommendable, provided an ophthalmoscopic examination permits one to infer that there is no extension of the growth into the interior of the eye, and that the presence of the eyeball does not interfere with the complete excision of the tumor by limiting the field of operation.

Where there is any suspicion of an extension into the orbit an exenteration should be performed.

The results of operations on recurring growths are not encouraging, but I think that an exenteration of the orbit may be at times beneficial, provided there are no grounds to suspect an intracranial extension.

I take pleasure in expressing my thanks to Dr. E. Lopez for the pathological specimen which forms the basis of this paper; to Dr. C. M. Desverinne for the use of his laboratory; and to Drs. Santos Fernandez, Montalvo, and Lopez for the use of their libraries.

No.	Author	Publication	Nature of tumor	Age	Durat.	Operative procedure	Results	Remarks
1	Blegny	Zodiacus medico-gallicus, p. 81 (quoted by Morgagni, see below).	Calcareous concretion in optic nerve.					
2	Morgagni	13th letter (quoted by Demarquay—Traité des tumeurs de l'orbite, Paris, 1860).	Cyst of optic nerve.					
3	Morgagni	13th letter.—Art. 6,(Demarquay)	Calcareous concretion in optic nerve.					
4	Scarpa	(Demarquay.)	Scirrho-cancerous (?) tumor of orbit originating in optic nerve					
5	Astley Cooper	Velpeau, Méd. opératoire, 2e edition. ii., 212.	Aneurism of central artery of the retina.					
6	Demours	Traité des maladies des yeux, i., 75.	Tubercular tumor of optic nerve.	36 yrs.	2 mos.			
7	Schaeffner	Recueil de thèses Allemandes. (Demarquay.)	Cancerous(?) degeneration of optic nerve.					
8	Paw	(Wardrop,—see below.)	Hydatid cyst of optic nerve.					
9	Heavyside	(Wardrop,—see below.)	Tumor of optic-nerve sheath.					In Mr. Heavyside's museum
10	Wardrop	Essays on the morbid anatomy of the eye. ii.,-1834.	Cancerous (?) degen. of opt. n.					
11	Manoir	(Demarquay.)	Cancerous (?) degen. of opt. n.					
12	Shayer	(Demarquay.)	Cancerous (?) degen. of opt. n.					
13	Eliason	(Demarquay.)	Cancerous (?) degen. of opt. n.					
14	Panizza	(Demarquay.)	Cancerous (?) degen. of opt. n.					
15	Middlemore	(Demarquay.)	Cancerous (?) degen. of opt. n.					
16	M. Roux	(Demarquay.)	Cancerous (?) degen. of opt. n.					
17	———	Schmidt's Jahrbuch i.,-352	Optic nerve tumor.		2 yrs.	Enucleation and extirpation of tumor.		
18	Lebert	Maladies cancereuses, p. 841.	Cancer (?) of optic nerve.					
19	Lebert	Ibid.	Cancer (?) of optic nerve.					
20	Lebert	Ibid.	Cancer (?) of optic nerue.					
21	Lebert	Ibid.	Cancer (?) of optic nerve.					
22	Lebert	Ibid.	Cancer (?) of optic nerve.					
23	Critchett	Med. Times and Gaz., 1832.	Fibrous tumor of optic nerve.		15 mos.	Extirp. of tum. with preserv. of eyeball.		
24	Schott	Controverse über die Nerven des Nabelstrangs, 1836, also Ed. Med. and Surg. Journ. xi.,274.	Fungus hæmatoides, connected with optic nerve.			Exenteration of orbit.	No recurrence 7 years later.	
25	Middlemore	Gaz. médicale, 1838.	Scirrhus (?) orig. in opt. n. sh.					
26	Gosselin	Bull. de la Soc. de Chirurg. viii., 112.	Several cases.					
27	Schmidler	Dict. des sc. méd., xxxv., 20.	Aneurism of central art. of ret.		Blind sev. yrs.			Princess of Baden.
28	Walter	Musée anat. (Demarquay.)	Calcareous plate in optic nerve.					

No.	Author	Publication	Nature of tumor	Age	Dur.	Operative procedure	Results	Remarks
29	WALTER	Bull. de Therap., 1844.-xxvii.,25.	Gelatiniform cancer (?) (myxoma (?)).	13 yrs.	4 yrs.	Extirp. of tum. and eyeball.		
30	MACKENZIE	Diseases of the Eye. 1844.	Fibrous tumor.		2 yrs.	Extirp. of tum. and eyeball.	No recurrence 20 months later.	
31	DIXON	Med. Times and Gaz. 1858. (Parisotti and Despagnet, see below.)	Gumma of optic nerve.					
32	BÖHM	Dublin Quart. Journ. of Med. Sc., Feb., 1847.	Fibrous tumor.	19 yrs.	13 yrs.			Death from pulmo. phthisis found at autopsy.
33	RITTERICH	Weitere Beiträge zur Vervolkommung der Augenheilkunst, 1861–57.	Sarcoma.	4 yrs.	3½ yrs.	Enucleation and extirpation of tumor.	Death with cerebr. sympts. 41 hrs. later.	Traumatism with forceps at birth. Tumor extended to chiasma.
34	SKOKALSKI	Ann. d'ocul., xlvi., 43.	Scirrho-cancerous tumor (?).	4 yrs.	4 yrs.	Enucleation and extirp. of tum.	Reccurence at end of 5 yrs, filling orbit.	Recurring tum. operated by other surgeon, purulent meningitis, death on 6th day, extension into intra cranial portion of nerve.
35	ROTHMUND (Jun)	Zeh. klin Monatsbl. 1863., 262.	Cystoid myxoma.	15 yrs.	13 yrs.	Enucleation and extirp. of tum.		
36	v. GRAEFE	Arch. f. Ophth., x., 1, 193.	Myxoma.	23 yrs.	2 yrs.	Enucleation and extirp. of tum.		
37	v. GRAEFE	Arch. f. Ophth., x., 1, 201.	Myxoma.	29 yrs.	7 yrs.	Enucleation and extirp. of tum.		
38	GALEZOWSKI	Thèse de Paris., 1865.	Fibroplastic tumors in optic n. and brain.					Found at an autopsy.
39	v. GRAEFE	Arch. f. Ophth., xii., 2–100.	Glioma.	6 yrs.	2 yrs.	Enucleation and extirp. of tum.	Death from purulent mening. (13th day.)	Exten. of tum. to incr. portion of both nerves. and to corp. str.
40	LIDDELL	N. Y. Med. Journ., 1866.	Neuroma (?).	20 yrs.	8 yrs.	Exenteratio orbitæ.	No recurrence after 14 months.	No microscopic examination.
41	SICHEL (fils).	Gaz. hebd., 1871.	Myxoma gelatinosum.	16 yrs.	2 yrs.	Enucleation and extirp. of tum.	No recurrence after 2 years.	
42	HORNER	Correspondenzbl. f. Schw. Aerzte, April 1, 1871.	Myxo-sarcoma of optic nerve.			Enucl. and extirp. of tumor.		
43	QUAGLINO	Ann. di Ottalm., i., 1, 27 and 3, 337, 1871.	Myxo-sarcoma.	24 yrs.	5 yrs.	Enucleation and extirp. of tum.	Death 6 months after opera. with cerebral symptoms.	Traumatism of 5 years before.
44	NARKIEWICZ-JODKO	Nagel's Jahresbericht., 1872, 363.	Myxoma.	11 yrs.		Enucleation and extirp. of tum.		

45	Aron Heyman	"De neuromate nervi optici." —Berlin, 1872.	True neuroma (?).	19 yrs.				
46	Krahm	Zeh. klin Monatsbl., 1872, 103.	Carcinoma (metastatic).	30 yrs.	8 mos.			Found at an autopsy. Both nerves presented tum. Carcin. tum. of both ovaries. Found at an autopsy.
47	Goldzieher	Arch. f. Ophth., xix., 3-125.	Fibroma myxomat.	10 yrs.		Enucleation and extirp. of tum.	No recurrence 1 year later.	
48	Goldzieher	Arch. f. Ophth., xix., 3-134.	Glioma with myxomat. deg.	4½ yrs.	6 mos.	Enucleation and extirp. of tum.		*Extension into retina.*
49	Goldzieher	Arch. f. Ophth., xix., 3-139.	Myxo-sarcoma.	4 yrs.		Enucleation and extirp. of tum.	Relapse in orbit 1 year later; 8 years later child blind of other eye and paralyzed.	
50	Steffan	Augenheilanstalt, Francfort, 1873 33.	Fibro-sarcoma.	30 yrs.		Enucleation and extirp. of tum.	No recurrence 15 months later.	
51	Perls & Loch	Arch. f. Ophth., xix., 2, 287, 1873	True neuroma (?).	9 yrs.	2 yrs.	Enucleation and extirp. of tum.	No recurrence 5 months later.	Traumatism 2 years before.
52	Knapp	Arch. f. Ophth. and Otol., iv., 323. 1874.	Carcinoma (?) of outer sheath of optic nerve (endothelioma ?).		3½ yrs.	Extirpation of tum. with preservation of the eyeball.	No relapse 8 months later. (Patient living and healthy, eye shrunken, painless. No relapse 1894.—H. K.)	S preserved. Pt. under observation for 3 yrs. before oper.
53	Knapp	Ophth. Soc. Heidelb, 1874. (Ann. d'Ocul., 1875.)	Fibro-sarcoma.	40 yrs.	3 yrs.	Extirpation of tum. with preservation of the eyeball.		
54	Dusausay	Bull. de la Soc. Anat. x., 1875.	Angiolithic sarcoma (Endothelial sarcoma ?).	50 yrs.	18 yrs.	Enucl. and extirp. of tumor.	Profuse suppur. Death w. cereb. sympts. (8 days).	
55	Christensen	Nagel's Jahresb., 1875, 386.	Tumor of optic nerve.	21 yrs.		Euncl. and extirp. of tumor.		Tumor occupied intevag. space
56	Knapp	Arch. of Ophth. and Otol. v., 132.	Large orb. sarcoma. Optic nerve affected secondarily.	38 yrs.		Removal of tumor w. preserv. of eyeball.	Death on 12th day fr. acute nephr.	*Extension into the interior of the eye along opt. n.* Metastases in liver and testicle. Croupous nephr.
57	Grüning	Arch. of Ophth. and Otol., v., 508.	Myxoma.	34 yrs.	14 yrs.	Extirp. of tumor w. preserv. of eyeball.	11 yrs. later pt. alive and eyeball still preserved (Knapp).	History of fall.
58	Neumann	Arch. of Heilk., xiii., 310 (Knapp's report in Trans. Internat Congress, Phila., Sept., 1876).	Sarcoma (Psamoma, Endothelial sarcoma ?)	20 yrs.	3 yrs.	Extirp. of tumor and eyeball.		Structure partly alveolar, partly compact.
59	Reich	Arch. of Ophth., xxii., 1, 103.	Endothelioma.	12 yrs.		Enuc. and extirp of tumor.		
60	Schott	Arch. of Ophth. and Otol., vi., 276.	Endothelioma (both nerves).	55 yrs.				Found at an autopsy.
61	Holmes	Arch. of Ophth. and Otol, vi., 299.	Myxo-fibroma.	10 yrs.	4 mos.	Removal of eyeball and tumor.		
62	Alt	Arch. f. Augenh., vii., 1, 46.	Endothelioma.	31 yrs.	6 yrs.	Extirp. of tum. w. preserv. of eyeball.	Enucleation subseq. necessary.	

No.	Author	Publication	Nature of tumor	Age	Durat.	Operative procedure	Results	Remarks
63	SCHOTT	Arch. f. Augenh., vii., 1, 81.	Glio-sarcoma.	3½ yrs.	2 yrs.	Removal of tum. w. preserv. of eyeball.	Enucl. subseq. necessary. Death on 7th day fr. pur. meningitis.	Chiasma and other nerve invaded.
64	BRAYLEY	Ophth. Hosp. Reps., ix., 11, 123.	Fibroma.	67 yrs.	2 yrs.	Enucl. and extirp. of tumor.		History of blow.
65	ARMAIGNAC	Journ. de méd. de Bordeaux, 1878.	Encephaloid of rèt. and opt. n. (glioma?).	26 mos.		Enucl. and extirp. of tumor.	3 removals and 3 recurrences.	*Involvement of the retina* seems primary.
66	FORSTER	Arch f. Ophth., xxiv., 2, 103.	Fibro-nuclear tumor.	15 yrs.	3 yrs.	Exenteratio orbitæ.	No recurrence 1½ yrs. later.	
67	PUFAHL	Breiträge z. prakt. Augenheilk., v. Hirschberg, 1878, iii., 63.	Sarcoma.	14 yrs.		Enucl. and extirp. of tumor.		
68	STRAWBRIDGE	Trans. Am. Ophth. Soc., 1878, 383.	Tumor of optic nerve (glioma?).	31 yrs.		Extirp. of tum. w. preserv. of eyeball.	Subseq. loss of globe. S. of other eyes subseq. lost w. cerebr. sympts.	
69	SATTLER	Arch. f. Ophth., xxiv., 3, 127	Tubercular tumor of optic nerve.	5 yrs.		Enucl. and extirp. of tumor.	Death 5 months later from tubercular meningitis.	*Extension into retina.*
70	LEBER	Arch. f. Ophth., xxv., 189.	Myxo-sarcoma.	6 yrs.	18 mos.	Enucl. and extirp. of tumor.		
71	LEBER	Arch. f. Ophth., xxv., 195.	Myxo-sarcoma.	4½ yrs.	3 yrs.	Enucl. and extirp. of tumor.	Death 9 days later w. mening. sympts.	Extirp. incomplete due to intra cranial extension. Invasion of chiasma, foci in other nerve.
72	LAQUEUR	Arch. f. Ophth., xxv., 241.	Myxo-sarcoma.	14 yrs.	1½ yrs.	Enucl. and extirp. of tumor.	No recurrence 15 mos. later.	
73	HIGGENS	Brit. Med. Journ., Oct. 18, 1879, 616.	Fibroma.	5 yrs.	2 yrs.	Enucl. and extirp. of tumor.		
74	KNAPP	Trans. Am. Ophth., Soc., 1879, 557.	Myxoma.	2 yrs.	2 yrs.	Extirp. of tumor. w. preserv. of globe.	Eyeball subseq. lost thro' phthisis 9 mos. later S. of other eye lost by desc. neuritis. (Propagation or symp. ophth.?)	
75	CHENANTAIS	Soc. anat. de Nantes, 1879.	Med. alv. neuroma (sarcoma?).	18 yrs.	3 mos.	Enucl. and extirp. of tumor.		
76	EVETSKY	Arch. f. Augenh., xii., 1, 16.	Endothelioma.		8 yrs.	Enucl. and extirp. of tumor.	Recurrence 8 mos. later, filling whole orbit.	
77	RAMPOLDI	Congrès internat. d' opht., Milan, 1881.	Glio-sarcoma.			Enucl. and extirp. of tumor. (Quaglino.)		
78	PONCET (Cluny)	Arch. d' opht., 1881, 616.	Fascicular myxoma.	16 yrs.	13 yrs.	Enucl. and extirp. of tumor. (de Wecker).		
79	TRILLAIS	Journ. de méd. de l' Ouest-Nantes, 1881, xv., 74.	Glioma (?) (Myxo-sarcoma?).	62 yrs.	7 yrs.	Enucl. and extirp. of tumor.	No recurrence 3 yrs. later.	
80	LEBER AND VOSSIUS	Arch. f. Ophth., xxviii., 3, 33, and 283.	Myxo-sarcoma.	2½ yrs.	1½ yrs.	Enucl. and extirp. of tumor.	No recurrence 3 yrs. later.	
81	LEBER AND VOSSIUS	Ibid.	Myxo-sarcoma.	8 yrs.	1 yr.	Enucl. and extirp. of tumor.	No recurrence 9 mos. later.	
82	v. RECKLINGHAUSEN	Ueber die multiple Fibrome der Haut, u. s. w. Festschrift, Berlin, 1882.	Angiolithic fibroma.					Tumor at Strasburg Institute.

83	Lawson	Royal London Ophth. Hosp. Reps., 1882, 296.	Sarcoma of optic nerve sheath.	65 yrs.		Enucl. and extirp. of tumor.	Recurrence 1 mo. later. Death 3 mos. later.	Metastases in bronch. and mesent. glds., pancreas, duodenum, spleen, and liver. Sec. deg. of r. kidney. Ant. ⅝ of brain surface covered w. coag. lymph. foci of softening.
84	Huc	Thèse de Paris, 1882.	Fibro-sarcoma.	7 yrs.	2 yrs.	Enucl. and extirp. of tumor.	Death on 9th day. Cerebr. sympts.	History of fall. Chiasma invaded.
85	Hulke	Royal London Ophth. Hosp. Reps., 1882.	False neuroma (Sarcoma?)	19 yrs.	6 yrs.	Enucl. and extirp. of tumor.		
86	Veron	Rec. d'opht., 1883, 32.	Myxo-fibroma.	18 yrs.	3 yrs.	Enucl. and extirp. of tumor.		
87	Peabody	N. Y. Med. Record, Feb., 1883.	Sarcoma.					Found at an autopsy. D. fr. mening.
88	Roux	Gaz. des hôp., Aout 6, 1884.	Myxomat. tumor.	13 yrs.	4 yrs.			Pathological specimen.
89	Johnson and Prudden	Arch. of Ophth., xiv., 2, 151.	Myxo-sarcoma.	12 yrs.	6 yrs.	Extirp. of tumor and eyeball.		
90	Parisotti & Despagnet	Rec. d'opht., 1884, 725.	Fibroma.	31 yrs.	20 yrs.	Enucl. and extirp. of ant. port. of tumor.	4 yrs. later remainder incr. in size: exenteratio orbitæ. Death 2 days later.	
91	Kunachowich	Med. Obezz., Moscow, 1885, xxiv., 293.	Myxoma.	18 yrs.	10 yrs.	Enucl. and extirp. of tumor.		
92	Vossius	Berlin. klin. Wochenschr., Mar. 30, 1885.	Myxo-sarcoma.	20 yrs.	1 yr.	Enucl. and extirp. of tumor.		
93	Brayley	Sem. Méd., Nov. 24, 1886.	Sarcoma.	42 yrs.		Enucl. and extirp. of tumor.		
94	Panas	Jocqs, Thèse de Paris, 1887.	Tumor of optic n. (Giant-celled tum. tubercular?).	59 yrs.	6 yrs.	Enucl. and extirp. of tumor.	No recurrence.	
95	Tillaux	Leçon clin. reç. par. M. Guillet, Gaz. des hôp., Fev. 17, 1887.	Primary tumor of optic nerve (Sarcoma).	40 yrs.	18 mos.	Enucl. and extirp. of tumor.		Jocqs, in his thesis, believes that if left, myxom. deg. would have set in.
96	Frothingham	Am. Med. Assoc. (Arch. d' opht., 1887, 480).	Sarcoma.					
97	Frothingham	Journ. Am. Med. Assoc., Dec. 10, 1887 (Arch. d' opht, 1888, 96).	Tumor of optic nerve.					
98	Frothingham	Ibid.	Tumor of optic n.					
99	Lawson	Roy. London Ophth. Hosp. Repts., xii., Jan., 1888.	Myxoma.					
100	Sutphen	Trans. Am. Ophth. Soc., 1889.	Sarcoma.	10 yrs.				

No.	Author	Publication	Nature of tumor	Age	Durat.	Operative procedure	Results	Remarks
101	CIRINCIONE	Giorn. di neuropatologia, vii., 2.	Tubercular tumor.					
102	AYRES	Am. Journal of Ophth., March, 1890.	Myxo-sarcoma.	12 yrs.				
103	AYRES	Ibid.	Sarcoma.	22 yrs.				
104	KESSLER	Weekbl. der Nederland Tijdsch. v. Geneeskunde, ii., 781, 1891.	Intravaginal sarcoma of optic nerve (fibrosarc w. myx. deg.).	5 yrs.	18 mos.	Enucl. and extirp. of tumor.		
105	GARNIER	Zeh. klin. Monatsb. f. Augenh., xxix., 208.	Myxo-sarcoma.					
106	DITTRICH	Prager Vierteljahrschr., xii. (quoted by Elschnig).	Multiple cancerous foci in opt. nerve as well as in brain and other cerebr. nerves.					
107	ELSCHNIG	Arch. of Ophth., xx., 3, 370.	Metast. carcinoma of intracr. port of opt. n. extending into orbitæ.					Carcinoma renis
108	KEYSER	Ann. of Ophth. and Otol., 1892	Glioma.	8 yrs.		Enucl. and extirp. of tumor.	Death on 5th day w. cereb. sympts.	
109	RÖHMER	Soc. franç. d'opht., Mai, 1892 (Ann. d'Oc., cvii., 354).	Telangiectatic tumor of optic nerve.	3½ yrs.		Extirp. of tumor w. preserv. of the eyeball.	No recurrence 18 mos. later.	
110	LAGRANGE	Congrès franç de chirurg, April, 1892 (Ann. d'Oc., cvii., 377).	Myxo-sarcoma.	13 yrs.	3 mos.	Extirpat. of tum. w. preserv. of eyeball.	No recurrence 6 mos. later.	
111	BOURGEOIS	Soc. d'opht. de Paris, Sept. 10, 1893.	Myxo-sarcoma of opt. n. and orbit.			Enucl. and extirp. of tumor.		
112	BRAUNSCHWEIG	Arch. f. Ophth., xxxix., 1 (Extr. of Arch of Ophth., xxiii., 3, 354).	Tumor of optic nerve.					
113	BRAUNSCHWEIG	Ibid.	Tumor of optic nerve.					
114	BRAUNSCHWEIG	Ibid.	Tumor of optic nerve.					
115	BRAUNSCHWEIG	Ibid.	Tumor of optic nerve.					
116	ROCKLIFFE	Trans. Ophth. Soc. of U. Kingd., xiii., 101.	Tumor of optic nerve.			Enucl. and extirp. of tumor.		
117	FINLAY	*This paper.*	Alveolar fibro-sarcoma.	55 yrs.	2 yrs.	Enucl. and extirp. of tumor. (Lopez).	Death 1 yr. later w. cereb. sympts. For some time pt. had been idiotic and blind of other eye which showed total opt. n. atrophy.	*Extension into interior of eye.* Structure partly compact, partly alveolar, and partly myxomatous.

NOTE.—In collecting these cases I have frequently been unable to cousult the original papers and have had to be contented with extracts; as we are, here in Cuba, unfortunately very scantily supplied with ophthalmological literature, I have mostly utilized those of the *Archives of Ophthalmology* and those of the *Annales d'Oculistique*. Dr. Jocqs's valuable thesis has also been of great help to me in this particular. In view of this I trust I will be pardoned any inaccuracy that may have crept in.—C. E. F.

TRAUMATIC PARALYSIS OF THE ABDUCENS NERVE.

By C. ZIMMERMANN, M.D., Milwaukee, Wis.

SINCE the publication of the articles of Purtscher and Friedenwald in these ARCHIVES (xxiii., No. 4), in which the authors collected the reported cases of traumatic paralysis of the abducens nerve (fifty-seven, with their own), two further cases of unilateral traumatic basal paralysis of the abducens have come to my notice.

CASE 1 is described by Professor A. Eulenburg (*Neurolog. Centralblatt*, 1894, No. 16, and Litteratur-Beilage, der *Deutsch. Medic. Woch.*, 1894, No. 14, p. 102):

A man, aged thirty-two, was stabbed with a knife in the right temple. A copious hemorrhage followed immediately and he became unconscious. After a few weeks complete paralysis of the right abducens nerve was ascertained, and a slight degree of right-sided ptosis, which disappeared later on; inconsiderable impairment of vision, headache, and vertigo. Eulenburg assumes that the blade must have injured the sixth nerve, where it is exposed to an isolated lesion, apart from the other (motor and sensitive) ocular nerves—*i.e.*, before its entrance into the superior orbital fissure, and where, in its course toward the basilar clivus, laterally and behind the dorsum sellae, it enters the cavernous sinus, and runs on the lateral side of the carotis interna. The ptosis may have been due to a slight lesion of the third nerve on its passage in the superior wall of the sinus.

CASE 2 came under my own observation:

April 26, 1894—Thosten Thomsen, a Norwegian ship-carpenter, aged forty-seven, was struck by a man with a capstan bar on the left side of his head, a few *mm* above a point midway between the external auditory meatus and the occipital protuberance. He went down-stairs to the cabin, but then he fell unconscious, and did not recover until the next day, while he was lying in the hospital. Right after the blow blood came out of the ear. Later he was told by the nurse that in the evening after the accident he was brought to the Emergency Hospital, and that he vomited blood. The next day he recovered consciousness, but felt sick, and suffered from diplopia and vertigo; could not raise his head; or, when raised, could not hold it up. April 27th he was transferred to the Marine Hospital, where he lay in bed for ten days. Dr. J. M. Allen sent him to me for an examination of his eyes (May 12th). I found complete paralysis of left abducens nerve. Homonymous diplopia, even in right field of fixation, from increased contraction of internal rectus. Pupillary reaction normal. V R = $\frac{1}{1}\frac{6}{8}$—V L = $\frac{1}{2}\frac{6}{0}$ with—0.75 D = $\frac{1}{1}\frac{6}{8}$. V F normal. No ophthalmoscopic changes. The other cranial nerves, especially the facial and acoustic, not affected.

Left ear: Ordinary voice at eight feet; watch not at all. T. F. better opposite the ear than at mastoid process. The posterior wall of meatus and the posterior half of *Mt* are covered by dried blood. This feels hard to the touch with the probe, which is painful. After removal of the blood on May 25th no fracture of the osseous canal can be detected, but the *Mt* shows a vertical line between umbo and posterior margin, apparently the scar of a rupture, through which the blood had escaped. Tinnitus, headache, and vertigo. He cannot balance himself well, when he walks, feels "topheavy," as he expresses it, even when the left eye is tied up. June 1st he had chills and had to stay in bed for a day. While I observed him until July 2d, and as long as he stayed in the hospital until August 17th, his condition did not change, and is still the same, as a letter of February 3d from Dr. F. Gudmundsen, of Detroit Harbor, Wis., where the patient now lives, informed me—*i.e.*, the paralysis is permanent.

In this case the primary paralysis of abducens was undoubtedly of basal origin, due to a transverse fracture of the apex of the petrous bone, tearing or lacerating the

nerve at this point in its course. The hemorrhages from ear and mouth directly after the blow corroborate this diagnosis. That the fracture must have been a transverse, not a longitudinal one, becomes clear from the facial and acoustic nerves not being involved, and from the action of the force on the same side of the head in the line of the sagittal suture. The impairment of hearing was not due to an affection of the acoustic nerve itself, but rather to a lesion of the labyrinth and the middle ear, into which an effusion of blood had occurred.

A CASE OF ANOMALOUS DIPLOPIA, DIVERGENT STRABISMUS WITH HOMONOMOUS DIPLOPIA; TWO "FIXATION-SPOTS" IN THE SAME EYE.

By N. DARRELL HARVEY, M.D., Providence, R. I.

SINCE hearing the report of Dr. Thomas of Philadelphia on "Three Cases of Strabismus with Anomalous Diplopia," made at the last meeting of the American Ophthalmological Society in Washington, I have had an opportunity of examining a case of the same class. Such cases are exceedingly rare, no mention of their occurrence being made in most of the leading text-books, so that they become of interest both upon this account, as also from the standpoint of treatment.

Besides these cases of Dr. Thomas, others have been described by Swanzy, Berry, Theobald, and Agnew.

The case I am about to report, although possessing many points of interest in common with those already reported, I think may yet present a few additional ones.

All the cases of Dr. Thomas were submitted to operation to rectify the deformity, and in none was orthophoria obtained. The present case has had no operation. The deformity has been so far removed, and a parallelism of the eyes apparently obtained by non-operative measures.

Although muscular equilibrium for all distances is not yet permanently established, orthophoria for near has been attained, and a similar condition for twenty feet, I think, will soon be found.

The gentlemen above referred to have suggested the

presence of an additional "fixation-spot" in the deviating eye as the only solution of the problem of anomalous diplopia, and I feel that the facts of the following case add to the support of this theory.

The history of the case is this:

Miss B. L., seventeen, came May 5, 1894, complaining of pain and occasional swelling about the right eye. She stated the right eye had always squinted, but the deviation was more marked at times. She never remembers having had "double vision." She does not mind the deformity much, but wants relief from pain.

S. P.—General health is good; divergent strabismus of the right eye; her family physician can find no cause, unless it be her eyes, to account for all her trouble.

V O D = $\frac{6}{7.5}$ accepts + 0.50.0° (centrally). V O S = $\frac{6}{7.5}$ accepts + 0.25.90°. Ophthalmometer O D 0.25.0 − 90 +; O S 10 − 90 +. Accom. good. Fundus normal. With both eyes open and right one in divergence, patient has single vision far and near. On covering the left eye, the right one fixes centrally, but immediately returns to its former condition of divergence as soon as the left one is free. Diplopia cannot be induced by any of the usual methods.

There is not binocular vision for near, as shown by the stereoscopic test, but whether it exists for distance cannot be certainly stated.

May 24, 1894.—Under homatropine: V O D = $\frac{6}{6}$ − w + 1.0° (centrally). V O S = $\frac{6}{6}$ − w + 0.25.90°. Ordered Gl. + 1.0° = O D + 0.25.90° O S for constant use, and avoid close use of eyes as much as possible.

June 4, 1894.—Headache and pain about eye greatly relieved; still some pain remaining. Has Sp. accom. = 0.50. V = $\frac{6}{6}$ a 0. To continue to wear glasses and exercise with pr. 12° base in, and endeavor to see "candle flame" at twenty feet double.

June 11, 1894.—Can now see "double" with vertical prism test and red glass. Eyes seem much straighter, and at times when the patient makes an effort there seems to be no real divergence. Under vertical diplopia test, the right eye being in divergence, she now has "homonymous diplopia," taking prism 3° b. out to produce horizontal equilibrium for distance, and pr. 6° b. out for near. With pr. 20° b. in, which corrects all movement of recovery of the right eye under the cover test she has the same

homonomous diplopia. Advised to try "finger exercise" for the interni, for five minutes three times a day.

July 31, 1894.—Headaches very much less; formerly daily, now only ten in five weeks. Same muscular condition. To continue exercise as before.

October 10, 1894.—Eye has remained very straight, but of late turns, and more especially when she is bodily fatigued.

S. P.—No divergence now for far or near under the "cover test." With vertical diplopia test and red glass the right eye immediately diverges slightly, to a less extent than before, but a wider homonymous diplopia occurs for distance requiring now pr. 8° b. out to correct. She now has horizontal equilibrium for near.

Dynamical condition : Dist., ab. = 20°, ad. = 36°.

Convergence to 2-inch : Near, ab. = 20°, ad. = 26°.

She states that the eye feels better with pr. b. out, so she was ordered Gl. + 1.0° pr. 4° b. out. for the right eye,—same Gl. for the left.

November 3, 1894.—Prism has given more relief. Advised to continue as she is now doing.

November 16, 1894.—Adduction stronger,—feels well enough to begin work in dry-goods store. Advised to wear the glasses constantly, and use the "finger exercise" every day and report again in one month or three weeks.

December 7, 1894.—Has felt so well that she has not used the exercise much. Eye keeps straight most of the time. Abduction and adduction about the same as before. Under vertical diplopia test and red glass, eye diverges but slightly, now needing pr. 12° b. out to correct diplopia. With the same test and pr. 12° b. in, which now corrects all movements of recovery on the part of the right eye under the "cover test," after some few attempts both eyes continue to fix centrally, and the patient obtains horizontal equilibrium of her muscles for distance, getting the normal projection.

Now to sum up: the principal points of interest about this case are:

1. The presence of two "fixation-spots" in the right eye. The one, a false fixation-spot, being an eccentric hypersensitive portion of the retina to the temporal side, used in preference to the central spot entirely at first, but, since treat-

ment was begun, only under conditions which disturb the newly acquired binocular vision. The other, the true macula lutea, at first disregarded, except when the right eye was used alone, but, later, after parallelism of the eyes was acquired, concerned in the production of binocular vision.

2. The relief from pain, the correction of the deformity, and the acquisition of binocular vision by the correction of the refractive error, and the systematic exercise of the power of adduction without any operative interference. Inasmuch as the presence of a false fixation-spot is admitted, the question arises as to what extent it was concerned in binocular vision. From the above facts, while the eye was in divergence, such a condition of single vision, it would seem, did not exist. The image perceived by the right eye was mentally suppressed when both eyes were open, and only appreciated when the eye was made to fix centrally. But although not actively engaged in the production of binocular vision, this spot, nevertheless, did have a closer relationship to the macula of the other eye than did the central point, acting as the standpoint from which projection was estimated.

Under the stimulation which the tendency for binocular vision received from the mechanical exercise of the weak adductive power as well as the increased action of the innervation centres, the deviating eye became straight, and this false fixation-spot has given place to the true macula lutea, and is now regarded only under conditions which disturb binocular vision, and then only at times. As this function becomes more and more firmly established, the presence of the false spot is more and more disregarded, until finally we shall see the central spot predominant and normal projection maintained under all conditions.

Another singular point about the case is that the patient with weak adduction still obtains relief from a prism base out. This, I think, must be accounted for on the ground that there is a spasmodic esophoria, and the relief obtained from the prism base out is analogous to that seen from wearing a myopic cylinder, where there is a spasmodic myopic astigmatism due to weak accommodative effort, the true refraction being one of hyperopic astigmatism.

May 5, 1895.—As a further addition to the history of this case, I would add the following: The patient, partly from the fact that she has been feeling so well, as also forgetfulness, has neglected to use the exercise. Still, the eyes continue straight—pain and discomfort all gone. Orthophoria near and for short intervals for distance under the vertical diplopia test. Ordered to persist in her exercise.

CONGENITAL, MONOLATERAL FISTULA OF THE LACHRYMAL SAC, SITUATED ABOVE AND PARALLEL WITH THE INFERIOR CANALICULUS.

By JOHN DUNN, M.D., Richmond, Va.

Mr. L., aged 20, exhibits the following anomalous congenital opening into the right lachrymal sac. The inferior punctum is normal in size and appearance, it opens into the canaliculus, which in turn allows the passage of a probe into the lachrymal sac of this side. Directly internal to the punctum is a narrow slit, about 1½ *mm* long, through which liquid, if injected, passes into the lachrymal sac and thence into the nose. This secondary canal is directly above and parallel to the inferior canaliculus, and opens by its own mouth into the sac.

There is no communication between this secondary canal and the canaliculus. Two needle probes can be passed at the same time, one through each canal, into the sac. No pathological discharge can be forced from either canal. Mr. L. says that to his knowledge he has never had any inflammation of this eye. He is, however, from time to time annoyed with the accumulation of tears in the corner of this eye. What causes this accumulation of tears is difficult to say. It may be that the internal end of this secondary canaliculus, as well as its walls, are unprovided with any elastic tissue, and thus offer no resistance to the regurgitation of tears from the sac during the contractile efforts of the sac walls, or it may be that the presence of this canaliculus has prevented the proper development of the structures lying above the walls of the inferior canaliculus.

In the case of Mr. L. there is no scar tissue about the secondary canaliculus nor any evidence that this anomalous condition is the result of inflammatory changes. Nor is there any reason for believing that intranasal conditions cause the epiphora.

(*Cf.* "Congenital, etc., Fistulæ of the Lachrymal Sacs., by C. A. Wood, Archives of Ophthal., vol. xxiii., Nos. 1 and 2.)

A SECOND ATTACK OF PAPILLITIS OCCURRING IN A CASE OF POST-NEURITIC ATROPHY OF THE OPTIC NERVES.[1]

By G. E. de SCHWEINITZ, M.D., and A. G. THOMSON, M.D., of Philadelphia.

THE following case occurred in the practice of Dr. Wharton Sinkler, in the Orthopedic Hospital and Infirmary for Nervous Diseases, and we are indebted to him for frequent opportunities of examining the patient.

H. M. presented himself in the Out-Patient Department of the hospital on February 9, 1891, complaining of a series of epileptic attacks. His mother had died of some nervous disease; one brother was insane; his own mental state was good, but he had suffered much headache. There was no history of traumatism or syphilis, and no evidence of disease other than the nervous disorder.

The first attack occurred in December, 1889, the second nine months later, the third the next day, the fourth three months later, the fifth four months later, the sixth three weeks before presenting himself for treatment and the seventh the day before he came to the dispensary. These seizures manifested themselves soon after breakfast, the convulsive movements involving first the left arm, then the leg, face, and finally, the whole body.

The ocular examination at that time revealed the following points: Pupils normal; optic discs of good color; slight tortuosity of the retinal veins; very full lymph sheaths. Vision (direct and indirect) was normal. Macular region + 1 D.

On May 28, 1892, his eyes were again carefully examined. The discs were now slightly hyperæmic, their upper and lower

[1] Paper read before the Section of Ophthalmology of the College of Physicians in Philadelphia, February 19, 1895.

edges veiled, the veins unusually full, and the lymph sheaths markedly infiltrated. Vision, accommodation, and pupillary reflexes were normal; there was slight esophoria, but no paralysis of any ocular muscle.

After remaining under treatment for some months, he sought advice at the Hospital of the University of Pennsylvania, and while there was examined (March 4, 1893) in the eye dispensary by Dr. Wallace, who found double optic neuritis.

The next ocular examination, March 15, 1893, was made at the Orthopedic Hospital, and the following note was recorded: "Well-marked double optic neuritis; both discs swollen + 3 D; many fresh hemorrhages on and near the swollen nerve-heads; form and red fields normal. Vision, right, $\frac{20}{15}$; left, $\frac{20}{20}$."

The patient was admitted to the hospital in the hope of obtaining relief from his violent headache, which was persistent, as well as from the epileptic attacks, which had continued.

One month later, his general condition still being very good, the swelling of the right disc had increased to + 4 D, and that of the left to + 6 D. The eyes wandered in under cover, but the muscles were not paretic, neither was there diplopia; vision and field of vision continued normal. The patient was trephined, April 13th, by Dr. W. W. Keen over the right motor centres, a portion of the bone being removed. It was believed that the tumor was localized, but that it was not removable.

June 12, 1893. The patient has had less headache since the operation than during the past year. Eye examination on that day as follows: O D vision $\frac{20}{40}$, O S $\frac{20}{200}$; apex of the swelling in O D + 4 D subsiding on the temporal side; apex in O S + 6 D. In each macula degenerative changes, closely resembling those seen in renal retinitis, have appeared. Urine negative.

August 14, 1892. The patient had spasm on awakening from sleep. The head was jerked backward, the left arm convulsed; unconsciousness followed. This has been the first return of his convulsive seizures since the operation.

August 28, 1893. The apex of the swelling of the right disc was + 1 D, of the left + 4 D; margins much clearer than at previous examination; the exudation in the macular region was more marked and now extended from the macula to the disc.

October 30, 1893. The centre of the right disc was + 1 D, its outlines were clearly defined, with marked pallor on the temporal side. The swelling of the left disc was + 2 D, its outlines were

appearing; the splotch of macular exudation was becoming atrophic and decreasing in size; the fundus on both sides, with the exceptions noted, was healthy. Vision, O D $\frac{20}{30}$, O S $\frac{20}{200}$. During all this time the patient's general condition continued to improve and he had returned to his usual work.

December 7, 1893. The vision of the right eye was normal, of the left $\frac{20}{200}$. The ophthalmoscopic appearances of typica post-neuritic atrophy were present, the atrophic pallor being chiefly noticeable in the temporal half of the right disc, while it was general in the left. In the macular region of each there was a patch of retino-choroiditis becoming atrophic upon the left side. The visual fields were normal in extent and the color perception perfect.

May 21, 1894.—The same conditions were present as at the last examination, the improved circulation in the nerve-heads being notable. Vision of the right eye $\frac{20}{20}$, of the left $\frac{20}{200}$.

During July the patient had one or two slight attacks, confined to the face, and complained of "a heavy feeling" in the arm.

Sept. 17*th.*—An attack occurred on the 8th of this month affecting the face and hands; since, there have been headache and pain over the right brow and behind the ear.

Early in November the patient was seized with violent pain in the head, associated with vomiting, and he noticed at once that vision in both eyes commenced to fail, the failure being somewhat gradual in its progress. There was weakness of the legs—not paralysis—and a sensation of general debility, confining him to bed until January 21, 1895, when he presented himself in the hospital, and the following note was made in regard to his eyes:

Movements of the eyes normal in extent in all directions, although when attempting to look directly forwards there is apparent divergence of the visual axes; there is no nystagmus; the pupils are dilated ad maximum, and fail to react to light or to attempts at convergence; there is absolute blindness. In the right eye the media are clear, the disc is swollen + 6 D (macula + 1 D), the swelling being of grayish color, and most marked above and below; the arteries are smaller than normal, the veins dark and somewhat tortuous, and carry white lines along their margins. In the left eye the disc is swollen + 6 D, the mound-shaped elevation being even more gray than upon the opposite side. The small patch of atrophic retino-choroiditis in the macula is still visible. The other portions of the retina appear normal.

In brief, then, we have the following conditions in this somewhat remarkable case, which has been observed for a period of four years: Focal epilepsy, creating the suspicion of intracranial tumor. At first there are no abnormal ocular symptoms except passive hyperæmia of the retinal circulation and infiltration of the lymph sheaths. One year later, the epilepsy continuing, there is distinct congestion of the optic disc, which, in a few months, develops into optic neuritis, and on the patient's return to the Orthopedic Hospital, two years after the primary examination, is evident as a well-marked double papillitis of the type ordinarily called "choked disc," without material disturbance in vision or the field of vision, and unassociated with abnormalities in the external ocular muscles or the pupillary reflexes.

From that time on, with the exception of decided increase in the papillitis and depreciation of vision in the left eye, the ocular conditions remained unchanged until the trephining, April 13, 1893.

Between three and four months subsequent to the operation, the ophthalmoscopic appearances of subsiding neuritis begin, and by December 7, 1893, or eight months after the operation, there is fully developed post-neuritic atrophy, with preservation of good vision in the right eye, and very moderate (one tenth of normal) vision in the left.

Nearly one year after this date the patient reappears with the return of headache, vomiting, and other symptoms of intracranial disease, from which he had been previously free, totally blind, and revealing to the ophthalmoscope well-marked double optic neuritis, the apex of the swelling being as high (namely, 6 D) as it had ever been in the primary attack.

Second attacks of optic neuritis in completely atrophied nerve-heads are exceedingly uncommon. Dr. Gowers,[1] commenting on cases of this character, refers to a boy, aged twelve, under the care of Dr. Hughlings Jackson, with double optic atrophy and absolute blindness from intracranial disease some years previously, in whom distinct double papillitis occurred in the atrophied discs, associated with symptoms of cerebral tumor.

[1] *Medical Ophthalmoscopy*, 3d ed., p. 56.

Even when the atrophy is partial, or absent, according to the same authority, and this certainly is our experience, two attacks of neuritis are rare. Dr. Gowers describes a patient who suffered from cerebral tubercle and died from an attack of tubercular meningitis. The neuritis caused by the former lesion disappeared, leaving partial atrophy, when the discs became swollen a second time as the result of the meningitis. The case reported this evening belongs to the examples of partial atrophy, or, rather, of post-neuritic atrophy with preservation of good vision, perfect upon the right side and one tenth of normal upon the left.

With the reappearance of the neuritis, associated with symptoms of renewed intracranial disease, there was blindness, affecting not only the eye which had previously possessed poor vision, but also the one which up to this time had not suffered depreciation of sight. It is interesting in this connection to recall an observation sometimes made, that after the subsidence of the neuritis without much impairment of vision, there may be partial or entire loss of sight from renewed intracranial mischief without marked change in the ophthalmoscopic appearances.[1] In this case, on the contrary, the loss of vision is associated with great change in the ophthalmoscopic appearance, viz., double optic neuritis.

Another interesting phenomenon in our case is the following: In March, 1893, although the papillitis of the left eye was considerable (the swelling being + 6 D), the vision was normal. Three months later the vision in that eye had sunk to one tenth of normal, while that upon the opposite side had fallen to one half, and each macula now presented appearances not unlike those seen in albuminuric retinitis. These macular changes increased in the left eye, but remained stationary, or practically so, upon the right side. Gradually the vision of this eye, namely, the right, improved to the normal standard, while upon the left side, the vision, after having sunk as low as $\frac{3}{200}$, regained the $\frac{20}{200}$ level and remained there without further change until the reappear-

[1] Gowers, *Medical Ophthalmoscopy*, second edition, Appendix, p. 292, Case 17.

ance of pronounced intracranial mischief, when the blindness became absolute, affecting equally the left and the right eye.

Ophthalmoscopic appearances closely resembling those seen in albuminuric retinitis have been noted not infrequently in connection with cerebral tumor, and the plate of Mr. Edmunds,[1] illustrating this phenomenon is a familiar picture. If we may judge from a number of clinical experiences, and at least two post-mortem examinations, when these macular changes are pronounced and the papillitis is accompanied with the appearance of considerable mechanical congestion, the tumor is likely, other things being equal, to be found in the cerebellum, a point that is dwelt upon by Mr. Marcus Gunn in his paper on changes in the macula.[2] The early blindness which accompanies the optic neuritis associated with cerebellar neoplasms, is a clinical fact too well established to need description here.

It seems likely that in this case the original lesion, probably a glioma, was situated beneath the area exposed by the surgeon's trephine; that in the course of the months which have elapsed since that operation and the present time this tumor has extended, the extension having been preceded by a period of relief, both from headache and other symptoms of intracranial mischief, and in this extension it is not unlikely that the cerebellum has become involved.[3]

[1] *Transactions of the Ophthalmological Society of the United Kingdom*, 1884.
[2] *Trans. Eighth International Ophthalmolog. Congress*, p. 79.
[3] J. Walter Park (*Annals of Ophthalmology and Otology*, January, 1895) describes an interesting case of acute middle-ear inflammation associated with two attacks of double optic neuritis, the interval between the attacks being a little more than a year. The condition of the optic nerves between the attacks is not stated. The patient made an excellent recovery in all respects, the final vision being $\frac{1}{10}$ in the right eye and $\frac{1}{10}$ in the left.

THE PARALLAX TEST FOR HETEROPHORIA.[1]

By D. A. DUANE, New York.

In the summer of 1889 I published in the *New York Medical Journal* a description of what I denominated the parallax test for insufficiencies. Since that time a continuous experience has convinced me that in this test we have a method which is not only among the most satisfactory of all that we possess, but also in some cases succeeds when the other tests fail or are self-contradictory. As the test is not generally known, and as the only detailed account of it appeared in a general and not a special medical journal, it seems not without utility to rehearse briefly the method of its performance and the principles upon which it is based.

To conduct the test, we place the patient in the primary position, with head erect and eyes directed straight forward or slightly below the horizontal plane (this latter especially in making the test for near points). The object of fixation, which should be twenty feet distant, may be a candle-flame, but preferably is a white spot, 1 or 2 *cm* in diameter, upon a dull black surface of some considerable extent. By this arrangement all danger of projecting the image upon a surface beyond is done away with, and the chance of a confusion with surrounding objects is prevented. The patient's gaze being directed fixedly at the spot, a card is placed before one eye, and passed alternately from that to the other,—the patient being at the same time asked whether the spot appears to move, and, if so, in what direction. If it remains perfectly stationary, there can have been no deviation behind the

[1] Paper read before New York Academy of Medicine.

card, and the position of fixation of both eyes is perfect. If, however, the spot moves, it must occupy a different position as seen by the two eyes; *i.e.*, there is really a diplopia present which our method of observation has unmasked. Thus if, on uncovering the left eye, the object (which was previously seen by the right eye and is now seen by the left) appears to move to the patient's left, there is really a homonymous diplopia (homonymous parallax), which differs from ordinary diplopia only in the fact that the two images are seen alternately instead of at the same time; if the object seems to move to the right, there is crossed diplopia (crossed parallax); if the object moves down, the eye must have been higher behind the screen (left hyperphoria, left parallax); and if the object moves up, the left eye must have been lower behind the screen (right hyperphoria, right parallax). In order to determine the amount of this alternate diplopia, we place prisms of the appropriate direction and strength before one eye until the movement is abolished. Thus, supposing that when the left eye was uncovered the object seemed to move down and to the left (homonymous and left parallax, indicating a condition of hyperesophoria), two prisms are placed before this eye, with their bases respectively down and out, and increased in strength until the movement has become nil. The strength of the prism having its base down will measure the degree of hyperphoria, and that of the prism having its base out will indicate the degree of esophoria present.

For near points the test is made in the same way, a small dot on a rather large card being employed, and the movement of the dot upon the card (and not of the card itself projected against some distant object) being observed.

Such being the nature of the test, and such the simple means by which it is carried out, it remains to state the reasons for employing it at all. This is requisite, for we have already a number of methods of measuring heterophoria, and the question may naturally be raised why we should multiply tests when enough exist already for the purpose in hand. The answer to this is that the complexity of the problem presenting itself for solution is often such

that it is desirable to get confirmatory evidence of every kind before deciding, and, moreover, the other tests, while indicating the kind of heterophoria that is present, not infrequently give an erroneous impression as to its amount. Hence it is useful to have an additional source of information to supplement and check those which we have already; and it only remains to inquire if this method that we have described answers the demands that we can rightly make of it.

Of any test thus put forward we must ask how far it fulfils the requirements of applicability, precision, accuracy, and utility. As regards the first requirement, I may say that in my hands the test has proved to be almost universally applicable. There are very few persons who cannot with a little patience on the part of the examiner be got to recognize the displacement of the image, and to indicate its direction with accuracy. If the displacement is not recognized at once, it will in most cases be noticed after the patient's attention has been called to the artificial parallax produced when a prism of 2° or 3° is placed before the eyes. That is, the prism by exaggerating the parallactic displacement has forced it upon the patient's attention, and has made him recognize in what it consists; and he is then able to say whether a similar movement does or does not exist when there is no prism before the eye.

As regards precision the test leaves nothing to be desired. A prism of $\frac{1}{2}$° suffices to produce or to neutralize quite a decided lateral parallax; and a vertical movement, corrected by a prism of even $\frac{1}{8}$°, is clearly noticeable.[1]

As regards accuracy, I have become convinced from the constancy of the results which the method affords that it is one of the most reliable of all that we have at our command. And this, in fact, constitutes its reason for existence; establishes its claim to be used with, and sometimes in preference to, the other methods of determining insuf-

[1] This precision makes it useful in determining whether a glass that the patient is wearing has or has not a prismatic element, and whether it is centered or not. If, for instance, a patient, who has no hyperphoria, shows, with his glass on, a vertical parallax, it proves either that the glass has a prismatic element in it, or is faultily adjusted (decentred) so as to produce a prismatic effect.

ficiency. A word upon this point will make this statement clear.

All the subjective tests for insufficiency depend in some way or other upon the production of diplopia. Usually this is effected by prisms, either applied roughly in the trial-frame, or with more precision in some one of the numerous varieties of phorometers. But the difficulty with the use of prisms has been that when two images of similar character are presented to the patient's notice, and he is required to state whether the two are or are not in the same vertical or horizontal line, he often unconsciously tries to bring them into line, and thus conceals an insufficiency which actually exists; or if the prisms are rotated, and the position of the images thus displaced, he instinctively tries to keep the images in line, and thus simulates an insufficiency which does not exist. This is often seen with the phorometer, and vitiates the findings of that otherwise most accurate instrument. Thus, when testing for near points, we are apt to find an exophoria, which increases almost indefinitely, although it is often quite certain that no such tendency to outward deviation actually exists. And while there are ways of obviating this objection that suggest themselves to the mind of the experienced examiner, they still constitute a source of uncertainty, particularly when the test is made for near points. Hence the attempt has been made to disassociate the images by giving one a different appearance from the other—*e.g.*, by converting a point of light into a luminous bar (Maddox rod), or into a blurred diffusion-circle (Stevens's spherical lens, with small aperture). But, as far as my experience goes, the impulse towards bringing two dissimilar images into line is almost as strong as when the images are alike. I believe that this is particularly the case with the Maddox rod. On account of this inaccuracy sometimes attaching to the subjective tests, some observers have preferred to rely solely upon objective methods, and have fallen back upon the old exclusion-test—*i.e.*, that in which the observer takes note of the deflection of one eye behind a screen, while the other eye is performing fixation. Now the parallax-test is

nothing but the subjective correlative of the objective exclusion-test, and is superior to the latter, both in being far more delicate and in admitting of precise measurement. Like the exclusion-test, it may be applied while the patient is looking in any direction whatever, and thus a difference in the amount of deflection in various parts of the field of fixation may be observed and estimated.[1] Moreover, the results are stabile, and when once the deflection is corrected, any further attempt at correction with prisms will produce a deflection in the opposite sense, so that there is not in this test, as in some of the others, a tendency on the part of the patient to overcome the prisms, and thus simulate an insufficiency which does not exist. In other words, the two images, being seen alternately, there is not the same impulse to force them together, as obtains in the other tests, where the two images are seen simultaneously. Hence it will often be found that when the phorometer indicates an insufficiency of the interni, or exophoria, of anywhere from 5° to 10° for near, the parallax test will show constantly a deviation outward of not more than 1°; and corresponding to this small deviation it will be seen that the eyes remain steady behind the screen in performing alternate fixation.

Having used the test now for eight years, and upon a great number of cases, I am thoroughly convinced of its utility, and desire to recommend it to those who busy themselves with the consideration of anomalies of the ocular muscles. And whatever may be thought of the rôle that these anomalies play in producing direct or reflex disturbances, it is at least important that the data upon which our judgment of this difficult question is based should be as precise and as free from error as possible.

[1] I have already (ARCHIVES OF OPHTHALMOLOGY, xxiii., p. 71) signalized the importance of observing this difference, which may give us a clue as to the nature and etiology of the heterophoria.

SYSTEMATIC REPORT ON THE PROGRESS OF OPHTHALMOLOGY IN THE THIRD QUARTER OF THE YEAR 1894.

By Dr. ST. BERNHEIMER, in Vienna; Prof. C. HORSTMANN, in Berlin; and Dr. P. SILEX, in Berlin;

WITH THE ASSISTANCE OF

Dr. S. M. Burnett, Washington; Dr. Dantone, Rome; Dr. Herrnheiser, Prague; Prof. Hirschmann, Charkow; Dr. P. von Mittelstädt, Metz; Dr. Sulzer, Geneva; Dr. Werner, Dublin; Dr. C. H. A. Westhoff, Amsterdam; Dr. Schiötz and Dr. Ole Bull, Christiania; Dr. R. Greeff, Berlin; Dr. Deus, Berlin; and others.

Translated by Dr. Ward A. Holden.

Sections I.–V. Reviewed by Dr. ST. BERNHEIMER.

I.—GENERAL OPHTHALMOLOGICAL LITERATURE.

TREATISES ON GENERAL, BIBLIOGRAPHICAL, AND HISTORICAL SUBJECTS. STATISTICAL REPORTS.

152. 35th Annual Report of the Hospital for Eye Diseases of the Netherlands. Utrecht, 1894.

153. Tscherning. *The Ophthalmological Works of Thomas Young*. Translated into French and edited by Tscherning. Preceded by a portrait of Young, a eulogy by Arago, and a preface by Javal. Pp. 248, with figures and 3 plates. Höst & Son, Copenhagen.

154. Bellarminoff. Report on the work done by the mobile ophthalmological departments. *Wratsch*, 1894, No. 28–29.

155. VAN LEERSUM. Vertical chirography. *Weekblad van het Nederlandsch Tijdschrift voor Geneeskunde*, ii., p. 377, 1894.

156. CZERMAK. *Operations on the Eye.* Parts 6 and 7. Carl Gerold, Vienna, 1894.

157. FICK. *Text-Book of Ophthalmology and Ophthalmoscopy.* With 157 figures, some colored, pp. 486. Veit & Co., Leipsic.

158. STANDISH, MYLES. Report of the ophthalmic division of the Carney Hospital, Boston, for 1893.

159. ARGYLL-ROBERTSON. The contributions of ophthalmology to general medicine (Presidential address). *Trans. Ophth. Soc. Un. K.*, xiv., p. 1.

In the Utrecht Hospital (152) 4221 patients were treated, 504 being in-patients, and 519 operations being done, 91 extractions, 50 enucleations, 89 tenotomies, and 6 advancements.

The ophthalmological works of Thomas Young (153) are not readily accessible, as the originals are scattered through the physiological transactions, and Peacock's book of the miscellaneous works of Thomas Young has not been extensively circulated. TSCHERNING's undertaking to render these works accessible to the general public is praiseworthy. May the wish expressed in the preface by Javal, that this book may be the starting-point for new labors in physiological optics, be fulfilled.

SULZER.

In various remote districts in Russia where physicians are few, oculists (154) are sent for the purpose of affording at least temporary relief and of performing operations. The number of patients so treated was 7691, and 1466 operations were made.

HIRSCHMANN.

VAN LEERSUM (155) regards the vertical handwriting as better in every respect than the slanting. It is easier to read, more quickly learned, better for the eyes, and does not necessitate unfavorable postures. WESTHOFF.

The 6th and 7th parts of CZERMAK's (156) excellent work have appeared. Like the preceding parts these are distinguished by the lucid treatment of the material in hand.

Notwithstanding the number of good text-books, new ones are constantly appearing without there being a palpable need for them. FICK's (157) book is short and clear, and contains very good figures, many of which are colored.

At the Carney Hospital (Dr. STANDISH) (158) there were

registered 1810 new cases in 1893. Fifteen extractions are tabulated, mostly by the combined method ; the others with a button-hole in the iris ; no loss. BURNETT.

This address (159) contains remarks on the following subjects : Errors of refraction and nervous disorders ; the surgery of the tendon ; cocaine ; antiseptics ; counter-irritation, etc.

WERNER.

II.—GENERAL PATHOLOGY, DIAGNOSIS, AND THERAPY.

160. BLOCK. A case of cyclopia. *Weekblad v. h. Nederlandsch Tijdschr. v. Geneeskunde*, pt. ii., p. 409, 1894.

161. BACH. On the micro-organisms of the conjunctival sac, their natural and artificial growth, and on the antiseptic value of eye-salves. *Graefe's Arch. f. Ophth.*, vol. xl., 3, pp. 130–220.

BLOCK (160) reports the case of a well-developed infant which showed only cephalic deformities and lived five hours after birth. The upper eyelid had no lachrymal point and was notched in the middle. The lid margins were provided with lanugo hairs. The lower lid evidently had been formed from the growing together of the right and left eyelids, and a notch showed the point of previous junction, and here lachrymal points were visible. When the lids were separated an eye not differing perceptibly from the normal was seen exactly in the middle of the square orbit.

WESTHOFF.

BACH (161) made a great number of experiments in relation to the presence of bacteria in the conjunctival sac. A conjunctiva of normal appearance was repeatedly tested for bacteria, and then the effect of winking and of the tears was studied, and he endeavored to find whether it was possible to render an infected conjunctival sac sterile, and what procedure would best accomplish it. For the latter purposes the disinfectant action of the eye-salves most in use was tested. The more important conclusions are as follows : 1. The mechanical cleansing of the conjunctival sac with the simultaneous irrigation with physiological salt solution is preferable to washing the sac with irritating antiseptics. 2. The cleansing of the conjuctival sac even when it appears normal is necessary before an operation. 3. Bandaging the eye favors the development of germs, and those present multiply. All microbes however do not find favorable conditions, and the influence of the tears must be taken into account. 4. Since cultures from the

conjunctival sac of an eye which had been bandaged two days, were often sterile, it is proven that the sac can be made sterile. The course of healing is generally but little dependent upon the quantity of germs in the sac, but depends rather upon the technique and the operator, the course of the operation, the patient himself, and the condition of his circulatory apparatus. 6. An infection of the wound in cataract extraction can scarcely occur if proper precautions are taken.

The bichloride-vaselin salve (1:3000), and the 2 % silver and copper salves even in combination with an aqueous menstruum like the tears, have strong disinfectant properties; the yellow salve, less. These salves are permanently and almost absolutely sterile. If the sac was not previously specially infected, it is in most cases possible to sterile the sac and even the lid margin by using bichloride-vaselin for twenty-four to forty-eight hours. If the plate shows innumerable colonies of staphylococci, the sac will not be rendered sterile even when the bichloride-vaselin has been used six to eight times in the forty-eight hours.

III.—INSTRUMENTS AND REMEDIES.

162. ROGMAN. Thioform in ocular therapeutics. *Flandre médic.*, Aug., 1894.

163. NICATI. Esthesiometry and photometry. A note in regard to the visual chart of the author. *Arch. d'opht.*, xiv., 5, p. 297.

164. GALEZOWSKI. Double spectacles with superimposed lenses for the use of aphakic patients and hyperopes. *Rec. d'opht.*, 1894, p. 525.

165. ANTONELLI. The Javal ophthalmometer employed for exophthalmometry and ophthalmostatometry. *Arch. d'opht.*, xiv., No. 9, 1894.

166. SIKLOSSY. On subconjunctival injections of bichloride. Report of a paper read before the twenty-seventh meeting of Hungarian physicians and natural scientists. *Wiener med. Wochenschr.*, 1894, No. 45.

167. DEUS. A new electric ophthalmoscope lamp. *Deutsche med. Wochenschr.*, 1894, No. 28.

168. PRINCE. The inclinometer in ophthalmology. *Ophth. Review*, xiii., p. 227.

169. LANDOLT. An ophthalmotrope. *Trans. Ophth. Soc. Un. K.*, xiv., p. 256.

170. MCGILLIVRAY. The therapeutic value of ice in ophthalmic surgery. *Ophth. Review*, xiii., p. 294.

171. BOKENHAM. On benzol-pseudo-tropeine or tropa-cocaine. *Trans. Oph. Soc. Un. K.*, xiv., p. 234.

172. HOWE, LUCIEN. Orthochromatic plates for photographing the interior of the eye. *Ibid.*, p. 251.

ROGMAN (162) has obtained favorable results in the treatment of purulent keratitis with thioform, which is used in the same way as iodoform. SULZER.

GALEZOWSKI (164) has devised a spectacle frame with a second clip allowing an accessory glass to be placed in the frames in front of the stationary glass. Presbyopic, hyperopic, and aphakic patients can thus dispense with the frequent changing of their glasses. SULZER.

ANTONELLI (165) used Javal's ophthalmometer for measuring the exophthalmus, the distance between the eyes, the difference in height, the deviation of one eye, and the extent and rapidity of nystagmic movements. The correct position of the ophthalmometer is obtained by means of a special apparatus, which is described in the original. In a case of pulsating exophthalmus he measured the extent of the exophthalmus, and found that even when the eye sank back in the orbit it still projected *2 mm* anterior to the other eye. In congenital height differences, which occur with asymmetry of the skull, the author found the lower eye almost invariably hyperopic and astigmatic, a condition which, in his opinion, is of importance in the development of squint. v. MITTELSTÄDT.

SIKLOSSY (166) has used subconjunctival injections of bichloride in fifty-six cases of acute blennorrhœa with corneal complications, and in numerous cases of acute trachoma with corneal ulcers and pannus, and has obtained quick and good results with this method, which is not so painful as it is said to be.
HERRNHEISER.

This is a graduated, flat, metal ring (168), with a strongly marked white line projecting towards the centre, and serving as an indicator. It can be used for finding the inclination of the shadow in retinoscopy, the position of the axis of a cylinder, the position of an obliquely elongated image, etc. WERNER.

This is a very simple and useful instrument (169) for demonstrating ocular movements, diplopia, etc. WERNER.

McGILLIVRAY (170) recommends *continuous* cold in the early stage of eye injuries by means of iced compresses for a day or two. The cold constricts the blood-vessels and so prevents exudations and diapedesis ; it lessens pain by preventing pressure on the nerves, and also by paralyzing them, and it also impairs the activity of pathogenic micro-organisms. WERNER.

BOKENHAM (171) finds that tropa-cocaine acts more rapidly than cocaine, but does not cause mydriasis, cycloplegia, nor vascular changes, and is less poisonous. WERNER.

HOWE (172) showed photographs taken by the method described by him in the *Trans. of the American Ophth. Soc.*, vol. iv., p. 568. Plates for the purpose should be orthochromatic and as rapid as possible. WERNER.

IV.—ANATOMY.

173. KESSLER. The perichoroidal space in its relation to the lymph circulation in the eye. *Oogheelkundige Verslagen en Bijbladen.* Utrecht, 1894, p. 199.

KESSLER (173) found that Fontana's spaces and the loose tissue between the ciliary muscle and sclera form the beginning of the perichoroidal space, which is a fairly large lymph space furnishing a passage for fluid from the anterior chamber toward the perichoroidea, Schlemm's canal, and Leber's plexus. In all eyes with subnormal tension, the perichoroidal space becomes larger and may extend back to the optic nerve. Eyes with cyclitis are favorable objects in which to study the chief directions of the lymph currents, after the leucocytes have been stained. If the perivascular sheaths of the venæ vorticosæ can be considered the passages of exit for the fluid in the perichoroidal space, the continuous movement of the eyes would be an excellent means of conducting the lymyh through the loose tissue of Tenon's capsule toward the orbital tissue from which it can be readily carried off. WESTHOFF.

V.—PHYSIOLGY.

174. TEPLIACHINE. Studies on the secretory nerves of the lachrymal gland. *Arch. d'opht.*, xiv., No. 7, 1894.

175. PARINAUD. The sensibility of the eye to spectral colors ; functions of the retinal elements and the visual purple. *Annal. d'Ocul.*, cxii., p. 228.

176. KOENIG. On the visual purple in man and its function in vision. *Sitzungber. der K. preuss. Akademie d. Wissenschr. zu Berlin*, p. 577.

177. SATTLER. Investigations on the question of the existence of an external accommodation from muscular pressure. *Graefe's Archiv*, xl., 3, p. 239.

178. v. HIPPEL. On total congenital color-blindness. *Festschrift for the 200th Anniversary of the University of Halle.*

179. HITSCHMANN. On the dream-life of the blind. *Zeitschr. f. Psych. u. Physiol. d. Sinnesorg.*, vii., parts 5 and 6, p. 387.

180. MARBE. On the doctrine of visual sensations resulting from successive excitations. *Inaug. Dissert.* Freiberg, 1893.

181. STERN, L. W. The perception of motion by means of the eye. *Zeitschr. f. Psych. u. Physiol. d. Sinnesorg.*, vii., parts 5 and 6.

182. STERN, L. W. The perception of changes in illumination. (Supplement), *ibid.*

183. v. HELMHOLTZ. *Handbook of Physiological Optics.* 2d ed. 8th part. Voss, Hamburg and Leipsic, 1894.

184. GRIFFITH. Criticism concerning recent views as to the secretory function of the ciliary body. *Ophth. Review*, xiii., p. 247.

TEPLIACHINE (174) reviews the views previously held in regard to the secretory nerves of the lachrymal glands and endeavors to solve the question by his own experiments, which led him to the following conclusions:

The view that the facial is the secretory nerve is untenable. The secretory fibres of the lachrymal and subcut. malæ nerves run intracranially in the trigeminus trunk and take part in the reflex secretion of tears and that caused by psychic influences. The normal and constant secretion of tears is not brought about reflexly by irritation of the anterior segment of the eye, and does not depend upon the secretory fibres of the lachrymal nerve, but is produced by the cervical sympathetic. The author observed two cases of complete unilateral facial paralysis in which no lachrymation could be produced reflexly, but there was also paralysis of the fifth nerve with anæsthesia of the cornea, conjunctiva and skin of the face. Notwithstanding this, the eye of the paralyzed side was more moist than the other, and the secretion of tears continued. v. MITTELSTÄDT.

PARINAUD (175) by means of a spectrum apparatus had made a

series of experiments on the perception of the spectral colors. The increased sensibility of the adapted retina differs with the different wave lengths. It is o for red, and gradually increases toward the blue end of the spectrum. The maximum of intensity in the spectrum for the adapted retina is at D, and for the unadapted eye is near E. The increased sensibility for a given wave length is only as regards light intensity, and not color perception.

KOENIG'S (176) careful experiments with monochromatic light and his mathematical calculations prove that different colors pass into different depths of the perceptive layers of the retina so that the different colors are not perceived in the same substance. Between the colors of shortest and longest wave length, there is a difference of 0.0786 *mm*, indicating that the perception of light of greater wave length takes place farther outward in the retina than the perception of light of shorter wave length. These facts do not agree with the color theories of Hering, Ebbinghaus, Donders, Wundt, and Franklin, while they are in accord with the Young-Helmholtz theory.

V. HIPPEL'S (178) careful examination of the eyes of a woman of twenty-seven with congenital total blindness furnishes a new support for the Hering theory.

Shortly after HELMHOLTZ'S (183) death, the eighth part of the second edition of his *Physiological Optics* appeared, which will be completed by A. Koenig. This part contains the doctrine of contrast sensations, of the different subjective sensations, of visual perceptions in general, and of ocular movements.

GRIFFITH (184) gives a detailed account of the structure of the ciliary region partly from the study of bleached sections. (He recommends bleaching the tissue *en bloc*, with a solution of chlorate of potash and hydrochloric acid, in order to avoid the brittleness of bleached sections.) He arrives at the conclusion that the aqueous humor is secreted by the plicated surfaces of the ciliary body and not by the glands of the ciliary body. True glands do exist in the pars non plicata, but contrary to Collins's opinion they are very few in number (two or three in a section). The complicated folding of the surface of the ciliary processes, combined with obliquity of section, may produce appearances like glands ; again, foldings of the epithelium produced by contraction of the ciliary muscle may be mistaken for them. There are no real glands in the ciliary region of albino rabbits, yet they

have aqueous humor. The few glands which do really exist in human eyes may probably control the amount of pigment in the eye. The theory of glandular hyperplasia in cyclitic membranes, as enunciated by Alt, and accepted by Collins, he considers absurd and contrary to general pathology. He looks upon these so-called gland proliferations as epithelial foldings due to cicatricial contractions, or as newly formed lymph channels congested with leucocytes. Against Nicati's view, that the aqueous humor is formed by exudation from the choroidal chorio-capillaris, which is contained between two impermeable membranes, the lamina vitrea internally and the intervascular membrane of Sattler, (tapetum) externally, the author points out that extensive choroidal disease does not alter the intraocular tension, while cyclitis does; he also asks, Is the membrane of Bruch really impermeable? And does the tapetum exist in man?

WERNER.

Sections VI.–XI. Reviewed by DR. HORSTMANN.

VI.—ANOMALIES OF REFRACTION AND ACCOMMODATION.

185. KOTOSCHOWSKY. Sciascopy. *Wajenno medic. Journ.*, Apr., 1894.

186. KATZ. On the determinations of refraction by means of sciascopy. *Arch. f. Augenheilk.*, xxix., p. 142.

187. BORDIER. The determination of the acuteness of vision in ametropic eyes by means of Badal's optometer. *Arch. d'opht.*, xiv., 9, p. 562.

* 188. MARTIN. On monolateral myopia. *Annal. d'Ocul.*, cxii., p. 1.

189. MASSELON. On nasal sclerectasia in myopia. *Annal. d'Ocul.*, cxii., p. 20.

190. HORI. A contribution to the operative treatment of myopia of high degree. *Arch. f. Augenheilk.*, xxix., p. 142.

191. FUKALA. The indications and contra-indications for operation in myopia of high degree. *Wiener klin. Wochenschr.*, No. 47, 1894.

192. ANDOGSKY and DOLGANOFF. Clinical notes on astigmatism and its correction by means of Javal's ophthalmometer. *Wratsch*, 1894, No. 36–37.

193. Risley. The results of treatment and the optical correction of ametropia in arresting the increase of myopia. Arch. of Ophth., xxiii., p. 247.

194. Southard. The modern eye. *The Refractionist*, June, 1894.

195. Smith, Priestley. On periodical testing of the eyesight in schools. *Trans. Ophth. Soc. Un. K.*, xiv., p. 220.

196. Wray. The eyesight of children. *Ibid.*, p. 228.

197. Clarke. The association of blepharitis and ametropia with analysis of 100 cases. *Ophth. Rev.*, xiii., p. 345.

Katz (186) calls attention to the sources of error in sciascopy, which are present both when the macular region is tested (involuntary accommodation, narrow pupil), and when peripheric portions of the fundus are tested (excavation), using the peripheric less curved portion of the cornea, etc., and also the fact that the shadow does not reverse at a neutral point but in an interval whose centre corresponds approximately to the far point of the examined eye. The greatest accuracy is obtained: 1st, by throwing the light in a direction which makes the smallest possible angle with the visual axis of the examined eye; 2d, by using as source of light a round sharply outlined surface; 3d, by dilating the pupil of the examined eye and by making the perforation in the mirror small. Hirschmann.

Bordier (187) shows that for determining the actual acuteness of vision in ametropic eyes, Badal's optometer is the best adapted instrument, and he gives the necessary procedures for cases of axial ametropia and ametropia of curvature.

v. Mittelstädt.

Martin (188) collected accurate clinical and ophthalmometric data of 180 cases of unilateral myopia, and criticised the theories of the production of myopia. Astigmatism, corneal inflammation, and trauma are important factors. Sulzer.

Nasal sclerectasia (189) was mentioned in Masselon's *Ophtalmoscopie Clinique*, 2d edition, 1891. Sulzer.

Hori (190) reports fifteen cases of myopia of high degree in which Schweigger in 1892–93 performed discission of the lens. The oldest patient was thirty-four. The myopia varied from 13–14 D. In three cases there was slight hyperopia after the operation, in three slight myopia, and in nine emmetropia. The acuteness of vision was more or less improved in all.

Fukala (191), who has now performed discission for myopia

in forty-four cases, recommends the operation only in persons under forty.

SOUTHARD's (194) paper is based upon an examination of 1300 ametropic eyes. Divided generally, there were 1017 cases of all forms of hyperopia, and 283 cases of all forms of myopia. The ages ranged from five years (1 %) to seventy years (1 %), the largest number (23 %) being at twenty years—that at ten years being 8 %; only 57.61 % had vision of $\frac{20}{20}$. There was simple H in 58 %, simple M in 8 %, and astigmatism of all kinds in 33 %. Other valuable tables too lengthy to abstract are given. BURNETT.

P. SMITH (195) advocates the yearly testing of sight, with Snellen's types, without glasses, by a master, who should report cases with V L $\frac{6}{18}$. This method involves no expense, and would detect all errors except hypermetropia, which of itself would cause symptoms sufficient to attract attention. WERNER.

WRAY (196) insists on surgeons examining the parents' refraction for prognosis and treatment of the children's eyes. Rapid disappearance of hypermetropia should be carefully noted. WERNER.

In 100 cases of blepharitis CLARKE (197) found 84 % of hypermetropia. Out of 200 eyes 194 were hypermetropic or astigmatic. In 96 cases of H. astig., 72 were not more than 0.5 D astigmatic. Average age seventeen. Only one patient was emmetropic, whereas in 100 eyes examined by W. Dodd, without disease, 20 % of emmetropes was found. WERNER.

VII.—LIDS.

198. ZEILENDORF. On congenital coloboma of the lid. *Wiener med. Wochenschr.*, 1894, No. 34.

199. KRISCHEWSKY. On the etiology of congenital coloboma of the lid. *Verf. d. Phys. med. Ges. zu Würzburg*, 1894, No. 5.

200. BERNHARDT. A contribution to the subject of the peculiar synchronous movement of the paretic upper lid in unilateral congenital ptosis. *Neurolog. Centralbl.*, 1894, No. 9.

201. HILLEMANNS. Peculiar synchronous movement of the upper lid of an eye with coloboma of the optic nerve. *Ibid.*, p. 388.

202. MÜLLER-KAUNBERG. Peculiar synchronous movement of an upper lid with ptosis, with movement of the lower jaw. *Jahresbericht d. Frohlich'schen Augenklinik in Berlin*, 1894.

203. STEPHENSON. Note upon a form of congenital trichiasis. *Trans. Opth. Soc. Un. K.*, xiv., p. 13.

204. WATSON, SPENCER. A new operation for trichiasis and distichiasis, with a case. *Ibid.*, p. 17.

ZEILENDORF (198) reports two cases of congenital coloboma of the lid. In the first case there was a V-shaped defect in the lower lid almost under the caruncle ; in the other case there was a similiar defect in each upper lid.

KRISCHEWSKY (199) believes that the congenital coloboma of the lids may be the consequence of the imperfect junction of the frontal and the superior maxillary process ; thus he would explain the triangular defects of the lower lid.

BERNHARDT'S patient (200), a boy of nineteen, with congenital right ptosis, raised the lid when the lower jaw was dropped or when it was moved to the left.

Similar cases were observed by HILLEMANNS (201) and MÜLLER-KAUNBERG (202).

Out of fourteen cases seen by the author (203) it was bilateral in nine. The displacement of the cilia, which occurs chiefly towards the inner canthus, is due to a fold of skin which rises up parallel to the lid margin, hides the intermarginal portion from view, without inverting it, and thus presses the eyelashes against the globe, causing irritation and corneal troubles. The author believes that defective development of the tarsus, and not any abnormality of the orbicularis is the cause of it.

(I have seen two instances of this condition in infants, and in one of these it disappeared under chloroform, a fact which would seem to support the second theory.—L. W.). WERNER.

This is a modification of WATSON'S (204) older operation. An oval flap of skin, above the lid margins, is left adherent by its under surface and then pushed through a " button-hole " and fastened in the intermarginal incision. WERNER.

VIII.—LACHRYMAL APPARATUS.

205. CUENOD. Two cases of dacryocystitis with ozæna. Bacteriological examination. *Arch. d'opht.*, xiv., 9, p. 495.

206. DIANOUX. Tumors of the lachrymal gland. *Annal. d'Ocul.*, cxii., p. 81.

CUENOD (205) in two patients with ozæna having acute dacryocystitis, found Lowenberg's ozæna bacillus in pure culture without other pyogenic microbes in the pus of the tear sac.

DIANOUX (206) saw within a short interval four cases of tumor of the lachrymal gland. The first was extirpated, recurred, and finally necessitated evisceration of the orbit. Six months after the last operation there had been no recurrence. The patient was thirty-six years old. In the second patient, aged twenty, there was recurrence fifteen months after the extirpation of a completely circumscribed tumor. In the other two cases the affection was tubercular. The two tumors removed in the second case had a similar structure, that of a polymorphous epithelioma.

SULZER.

IX.—MUSCLES AND NERVES.

207. SCHMIDT-RIMPLER. On binocular vision in squinting individuals before and after operation. *Deutsche med. Wochenschr.*, 1894, No. 44.

208. LANDOLT. On strabotomy. *Arch. d'opht.*, xiv., 8, p. 474.

209. PANAS. Paralyses of the motor muscles of the eye after lateral pressure on the skull. *Ibid.*, p. 465.

210. VALK. The dynamics or power of the interni. *Med. Record*, July 21, 1894.

211. HALL, GEO. P. A contribution to the study of the insufficiencies of the ocular muscles, with measures directed to their relief. *Trans. Texas State Med. Soc.*, 1893.

212. FROST. Nystagmus in a man who has worked in a bad light for eighteen years. *Trans. Ophth. Soc. Un. K.*, xiv., p. 245.

213. THOMAS, C. H. Three cases of strabismus with anomalous diplopia. An original and an acquired fixation spot in the same eye. *Ophth. Review*, xiii., p. 281.

214. WILLIAMS, R. The operation of advancement in squint. *Ibid.*, p. 377.

215. TAYLOR, JOHNSON. A case of probable disease of the lenticular ganglion. *Ibid.*, p. 290.

According to SCHMIDT-RIMPLER (207) a squinting individual ordinarily only sees the image of the eye to which he directs his attention. It is therefore advantageous to practise the squinting eye before the operation in order to facilitate binocular vision after the operation.

LANDOLT (208) on the ground of increased experience recommends, as before, advancement as the chief procedure for the correction of squint. He makes this operation, in cases of insuf-

ficiency and ordinary squint, on one eye or both, and when necessary with resection of a portion of the tendon. Only in cases of excessive deviation does he make a careful tenotomy in addition. The disadvantages of tenotomy—restriction of mobility, sinking of the caruncle, and protrusion of the ball—are impossible in advancement which corrects the squint in the entire field of fixation. An excessive effect need not be feared, nor a vertical deviation, nor a meridional torsion. v. MITTELSTÄDT.

In a man whose skull had been compressed laterally, probably causing fracture of both petrous bones and the sphenoid, PANAS (209) found paralyses of all the ocular muscles the right auditory and the left facial nerve. There was bleeding from the ears and nose immediately after the operation. Later there were severe headache and a sero-purulent discharge from the right ear, and two weeks after the injury the paralyses appeared. By literal compression of the head of a corpse Panas was able to produce a double fracture of the temporal bone which would have explained the symptoms found. In another experiment, by compressing the skull in the temporal region, he obtained a basal fracture of the middle fossa which extended toward the sphenoidal fissure, and passed through the outer wall of the left orbit and under the frontal portion of the sphenoid and the optic foramen on the right side. The temporal bones were simply displaced. In both experiments, the roof of the skull remained uninjured. Panas reports also a case corresponding to the second experiment, in which after a fall on the head there were hemorrhage from the left ear, bilateral subconjunctival hemorrhage, and paralysis of the abducens and optic nerves of the left side. Panas concludes that the majority of the ocular muscle paralyses after skull injuries are due to fracture of the base. The abducens is the nerve most frequently affected because of its nearness to the bone. The paralysis is due either to direct injury of the nerves, or to the presence of blood, or inflammatory exudation. v. MITTELSTÄDT.

VALK (210) has found, as the result of his experience and examinations in more than 50% of his asthenopic cases, an excess of power in the externi. He places but little reliance upon those methods of testing for insufficiency in which one retinal image is reduced by any means. He relies on the old prism test for ascertaining the actual power of the muscle—exercise has proved useful only in weakness of the interni. Where the insufficiency is well marked he does a partial tenotomy. BURNETT.

HALL (211) reports a number of cases where the lack of muscular balance was entirely independent of any refractive error; he has used gymnastic exercise, prisms, and tenotomies, all with success, in relieving the faulty condition. BURNETT.

FROST'S (212) patient worked at night washing cabs in a badly lighted yard. V = $\frac{6}{6}$. Nystagmus occurred on raising eyes above the horizontal line. Slight improvement on cessation of work. WERNER.

THOMAS (213) records two cases of divergent strabismus, in one of which there was *homonymous* diplopia before operation, while after it the diplopia agreed with the position of the eyes, V = $\frac{6}{6}$; H = 0.5 D. In the second case after operation, which diminished the amount of divergence, *homonymous* diplopia appeared. The last case, one of convergence, showed *crossed* diplopia after incomplete correction by operation. In all cases there was central fixation with each eye separately. WERNER.

WILLIAMS (214) makes a horizontal incision in the conjunctiva over the muscle, and the suture is passed through the muscle from side to side at right angles to the direction of its fibres. WERNER.

TAYLOR'S (215) patient, a woman, aged forty-nine, with — 16 D, developed rather quickly dilatation and immobility of left pupil, with some proptosis and a little haziness of vision; there was neuralgic pain about the eye and down the nose. She had suffered some years before from pain and loss of power in one arm. The author accounted for the ocular condition by assuming that orbital cellulitis was present and damaged the lenticular ganglion. WERNER.

X.—ORBIT AND NEIGHBORING CAVATIES.

216. MITVALSKY. On orbital thrombo-phlebitis. *Prager med. Wochenschr.*, 1894, No. 45.

217. BOERMA. A case of symmetrical lymphoma. *Graefe's Archiv*, xl., 4, p. 219.

218. MARTIN. Orbital tumors caused by frontal-sinus disease. *Annal. d'Ocul.*, cxii., p. 182.

219. BERGER. Varieties of the wall of the optic foramen. *Arch. d'opht.*, xiv., 9, p. 545.

220. GRIFFITH. Some cases of orbital growths. *Trans. Ophth. Soc. Un. K.*, xiv., p. 173.

221. SNELL. Congenital cysts of the eyelids associated with anophthalmus or microphthalmus. *Ibid.*, p. 190.

222. CLARKE. Pulsating exophthalmus (congenital). *Ibid.*, p. 202.

223. GREEN, E. C. A case of exostosis of the orbit; operation. *Ibid.*, p. 186.

224. WILLIAMS, R. Case of rapidly growing sarcoma of the orbit. *Ibid.*, p. 186.

225. SNELL, S. Rapidly growing sarcoma in a child involving both orbits with secondary growths. *Ibid.*, p. 160.

226. SNELL, S. Osteoma of the orbit. *Ibid.*, p. 169.

227. POWER, H. Case of microcephalus and proptosis. *Ibid.*, p. 312.

228. STEPHENSON, S. Cellulitis following Mules's operation. *Ophth. Review*, xiii., p. 286.

MITVALSKY (216) examined two cases of orbital thrombo-phlebitis clinically, anatomically, and bacteriologically. The first case, in a man of fifty-six, was due to a gangrenous amygdalitis. Anatomically there was found first a hemorrhagic inflammation of the orbital veins, and later an œdema of the orbital connective tissue. In the fresh venous thrombus and also in the sections the Fraenkel-Weichselbaum diplococcus was found. In the second case the staphylococcus pyogenes was the cause of the infection.

HERRNHEISER.

BOERMA (217), in a woman of sixty-three, observed symmetrical orbital tumors pressing the lids and conjunctiva forward, which could be pressed back to a certain degree beneath the orbital margin. The tumors were removed with preservation of the ball, and proved to be lymphomata of the orbit.

Following empyema of the frontal sinus, MARTIN (218) observed swelling of the neighboring orbital tissue and almost complete loss of sight, with exophthalmus and displacement of the ball outward and downward. Besides the subcutaneous collection of pus there was a protrusion of the orbital wall of the frontal sinus. Catheterization and antiseptic injections cured the case completely, and the author does not favor trephining the sinus.

SULZER.

BERGER (219) gives drawings of various frontal sections through the optic foramen, showing the varying thickness of the wall of the sphenoidal sinus adjacent to the optic nerve. In

places it is very thin on each side, again thin on one side and thick on the other. At times the wall is broken so that the mucosa of the sinus must lie in contact with the sheath of the optic nerve, and thus is to be explained the affection of the optic nerve in diseases of the sinus. In 1888 Berger had collected twenty-three such cases verified by autopsy. Of the other neighboring cavities, only the ethmoid could also play a similar rôle in the production of optic-nerve trouble. v. MITTELSTÄDT.

GRIFFITH (220) records the following cases: 1. Fibro-sarcoma in a man aged thirty-five, removed without recurrence. 2. Exostosis in a girl aged fifteen. 3. Symmetrical orbital tumors, with motor and sensory paralyses of orbital region, improving under treatment. 4. Probable fibrous tumor; and, finally, two cases of malignant sarcoma, with fatal result after operation, which consisted in circular incision round orbital margin, and removal of contents of orbit. WERNER.

SNELL (221) records three cases, two occurring in the lower lid with apparent anophthalmus, and one in the upper lid with microphthalmus. In the last case, the child vomited when the cyst was tapped; it pulsated synchronously with the anterior fontanelle. Chemical examination of the fluid showed that it was not cerebro-spinal. All three cases recovered, the two first after removal of portion of the cysts, and the last after three tappings. WERNER.

In CLARKE's case (222), the birth of the child was normal. In addition to the pulsation there was an intermittent blowing murmur. Optic disc atrophied, arteries small, veins dilated. The president mentioned two traumatic cases cured by electrolysis. WERNER.

In GREEN's (223) case the exostosis was removed by the electro-motor drill in about seven minutes, with success. WERNER.

In WILLIAMS's (224) case, a man aged thirty-three, death took place within two months after the onset of the symptoms. When the eye was enucleated no distinct tumor could be felt in the orbit, but twenty-four hours later the growth had filled it up. WERNER.

SNELL's (225) patient was aged four. A diffuse periosteal sarcoma affected the frontal bone, involving both orbits. WERNER.

In SNELL's (226) case, a girl aged twenty-five, there was an osteoma of the inner wall of the orbit, and another in the nostril, both of which were easily removed, and were probably unconnected. WERNER.

In POWER's (227) case the birth was normal, but the child lived only thirty days. The vertex and frontal region were drawn out into a blunt cone. The eyes projected forwards, owing to shallowness of the orbit, which was only 2.5 *cm* in depth. The roof was almost vertical, and the optic nerve ascended in contact with it.

WERNER.

Out of 30 cases in which STEPHENSON (228) did evisceration, with insertion of an artificial vitreous, this was the only case in which severe cellulitis occurred. The glass ball was removed, and the stump subsequently enucleated; the sclera was found to have undergone extensive hyperplastic changes. WERNER.

XI.—CONJUNCTIVA, CORNEA, ANTERIOR CHAMBER, AND SCLERA.

229. VENNEMANN. The surgical treatment of granulations. *Arch. d'opht.*, xiv., 7, p. 413.

230. GLEISER. The statistics of trachoma. *Annal. d. kais. Charkow 'schen Universität*, 1894.

231. BLAAUW. Some considerations on the nature and treatment of trachoma. *Inaug. Dissert.*, Amsterdam, 1894.

232. TRUC. The general character and contagiousness of trachoma in the region of Montpellier. *Annal. d'Ocul.*, cxi., p. 328.

233. KRUCH and FUMAGALLI. Amyloid degeneration of the conjunctiva. *Annal. d'Ocul.*, cxii., p. 39.

234. SAULAY. Note on conjunctival papilloma. *Rec. d'opht.*, 1894, p. 543.

235. SCHIRMER. Cure of a cicatricial pterygium by corneal transplantation. *Deutsche. med. Wochenschr.*, 1894, No. 37.

236. WEYMAN. Conjunctival polypi and their relation to malignancy. *The Ophth. Record*, Sept., 1894.

237. HOBBS. The treatment of pterygia with the galvanocautery. *Journ. Amer. Med. Assoc.*, Sept. 15, 1894.

238. JESSOP. Case of sloughing of both lids and the eyeball in an infant, with subsequent complete cicatrization. *Trans. Ophth. Soc. Un. K.*, xiv., p. 22.

239. HUTCHINSON. On school ophthalmia. *Ibid.*, p. 35.

240. RIDLEY. Some points on the etiology of trachoma. *Ibid.*, p. 24.

According to VENNEMANN (229) the follicles in trachoma are innocent and may disappear of their own accord, but as cicatricial shrinking follows in the hypertrophied and degenerated conjunctiva the surgical methods of treatment are not rational. The author therefore used the old procedure of Peters-Keining. After the use of cocaine the epithelium of the conjunctiva is removed with Demarres' scarificator, the conjunctiva is irrigated with 1:500 bichloride solution, and, the following day, rubbed with cotton which has been dipped in this solution. The third day a croupous membrane forms, which is a favorable sign. The results were good. Relapses occurred, but the treatment is not disagreeable and the patient readily consents to a second operation.

v. MITTELSTÄDT.

The total number of trachoma cases treated in the Charkow clinic (230) in the years 1867–1892 was 21,218, or 13 % of all the eye cases seen. The percentage of trachoma cases increased regularly from 7.8 % in 1867 to 16.4 % in 1892. A particular racial predisposition could not be made out. The percentage of severe, neglected, and complicated cases has steadily decreased from 59.2 % to 42.5 %. In the statistics of blindness for the years 1869–1873 trachoma was noted as the cause in 6 %.

HIRSCHMANN.

According to BLAAUW (231) trachoma is not contagious, but develops where hygienic conditions are unfavorable. A specific microbe has not been demonstrated and it is not probable that such exists. In typical trachoma there is no mucus and no pus. It is chiefly found in children under six. It is always chronic, and cases do not require isolation. WESTHOFF.

TRUC (232) sees in his clinic one hundred times more trachoma patients than in his private practice. Almost all the clinic cases are scrofulous. The purulent variety is rare, and the ordinary dry form is benign.

KRUCH and FUMAGALLI (233) came to the following conclusions. Amyloid degeneration of the conjunctiva is a local condition not dependent upon constitutional causes. The local excision of the diseased tissue improves the condition and may cure it, even when the excision is not complete. The amyloid substance is formed chiefly at the expense of the subconjunctival connective tissue, and does not originate from the vessels. It is probably of parasitic origin. SULZER.

In a case of large cicatricial pterygium involving two thirds of

the cornea, complicated with extensive symblepharon, SCHIRMER (235) detached the pteryguim from the cornea and detached the conjunctival folds and cicatricial bands from the sclera. The defect above the sclera was covered with conjunctiva, and the denuded corneal surface was scraped smooth with a sharp spoon and filled in with flaps from the surface of a rabbit's cornea, which remained opposed and healed there. The mobility of the ball was preserved and only a small corneal leucoma remained.

JESSOP'S (238) patient was aged nine months and had ophthalmia neonatorum. WERNER.

HUTCHINSON (239) discusses at some length the question of the nature of a mild form of ophthalmia with development of follicles, and in a very few instances leading to granular ophthalmia. Contrary to the opinion of nearly all present who took part in the discussion, he believed this form of ophthalmia to be the precursor of trachoma (in no case was there any pannus), and as such required stringent observation, and regulation as to isolation, etc. Most of the speakers looked upon the presence of small follicles as a very common condition in children. Stephenson found them in 94 per cent. of children in schools in which there was no ophthalmia, and he was disposed to regard them as normal. Doyne thought that the author's cases were cases of muco-purulent conjunctivitis in subjects whose lids were in a follicular condition. WERNER.

RIDLEY (240) in a very interesting paper describes the histology of the normal conjunctiva of the fornix, and also of the same part when affected with trachoma. Foa's solution was used as a fixing agent. The normal conjunctiva possesses stratified epithelium, the cells being columnar below, then rounded, and finally squamous on the surface ; there are some goblet cells near the surface. The folded condition at the fornix produces on section the appearances described as Krause's glands. There is a variable amount of diffusely scattered lymphoid tissue, which also exists under other mucous membranes, especially around openings. In all forms of conjunctivitis this lymphoid or "sentinel" tissue is increased ; this increase is not a disease, but evidence of the reaction of the organism to an injurious agent.

The lymphoid tissue in trachoma differs from the same tissue in other diseases only in its amount and arrangement. It occurs as a diffuse infiltration of the subconjunctival connective tissue, and in the shape of round packets, occasionally encapsuled,

covered only by epithelium, in which it causes projections and furrows. These lymph follicles resemble infective granulomata and contain epithelioid cells, but no giant-cells ; they differ, moreover, from tubercle in containing healthy blood-vessels.

According to the above, granular ophthalmia is not necessarily due to the same cause in every case, but there may be a specific cause in certain cases. The epithelium is probably the seat of the irritant, and improvement takes place on destroying the epithelium. According to the author's investigations, the normal conjunctival sac contains the same micro-organisms as it does when trachoma is present. The changes in the epithelium include increased proliferation, deepening of the folds, so as, in some places, to form cysts, and increase in the number of goblet cells. Many of these so-called goblet cells are not really such, for they lie in the deepest layers, often parallel to the surface, and they stain differently : they may be protozoa. Amongst other things, the slight contagiousness of granular ophthalmia is against its being of bacterial origin. WERNER.

241. FUCHS. On keratoplasty. *Wiener klin. Wochenschr.*, 1894, No. 45.

242. CRITCHETT. A case of conical cornea treated by galvano-cautery without perforation of the cornea, and subsequent small iridectomy. *Trans. Ophth. Soc. Un. K.*, xiv., p. 73.

243. GRIFFITH. Blood-staining of the cornea. *Ibid.*, p. 73.

244. COWELL and GRIFFITH. A case of filamentous keratitis. *Ibid.*, p. 76.

245. SCOTT, KENNETH. A new method of treatment for vascularized corneæ. *Ophth. Review*, xiii., p. 348.

FUCHS (241) has done total keratoplasty in 30 cases ; 2 with parenchymatous keratitis, 12 with true superficial corneal scars with adhesions of the iris, 8 extensive total ectatic scars from prolapse of the iris ; 14 times he used a rabbit's cornea, 11 times that of an enucleated human eye, and several times the cornea of dogs or of fresh still-born infants. In 11 of the 30 cases the transplanted piece of cornea did not adhere. In no case did the transplanted piece remain transparent, it becoming opaque, as a rule, after a few days.

In CRITCHETT's case (242) V was improved from Jaeger 8 at 3 inches to Jaeger 2 at 10 inches and $\frac{6}{18}$ — 9 Cyl.

WERNER.

GRIFFITH'S (243) patient had discission performed; three months later there was pain, high tension, hyphæma, and smoky cloudiness of the cornea, except at periphery. On section, the substantia propria of the cornea was studded with small, round, yellow granules, highly refractive, insoluble in acids, alkalies, ether, alcohol, etc., stained only by eosin- and iodin- or methyl-green. As in cases recorded by Lawford and Treacher Collins, there was high tension with the hyphæma, and also detachment of the retina. WERNER.

This case (244) presented the usual twisted filaments with bulbous extremities attached to the cornea. The bulbs stained with fluorescin. WERNER.

SCOTT (245), with the aid of a lens, slits open the corneal vessels with a Graefe's knife. He finds this much better than the cautery. WERNER.

246. FAGE. Immediate and late results of scleral sutures. *Annal. d'Ocul.*, cxii., p. 262.

247. COLLINS, E. T. Case of a patient with congenital excess of pigment in the uveal tract, and pigmentation of the sclerotic in one eye, which late in life became the seat of a melanotic sarcoma. *Trans. Ophth. Soc. Unit. King.*, xiv., p. 197.

FAGE (246), after a considerable experience in suturing scleral wounds with fine catgut, recommends the procedure. The late occurrence of irido-cyclitis, when the eye is apparently cured, is seen after injuries in which septic bodies have entered the vitreous. SULZER.

248. SNELLEN. Descemetitis. *Ophth. Review*, xiii., p. 259.

249. COPPEY. A case of filaria in the anterior chamber of a human eye. *Arch. d'opht.*, xiv., 9, p. 557.

SNELLEN (248) refers to the various views as to the nature of this affection, *e.g.*, a disease *sui generis*, serous iritis, serous cyclitis, catarrhal inflammation of the glands of the ciliary body; he then records three cases in which there was k. punctata, with slight circumcorneal injection, and no other symptoms of iritis, except synechiæ, where dots of lymph formed on the iris. In each case paracentesis enables the author to examine a dot, which he found to consist chiefly of short bacilli, showing, as he believed, that this was a disease *sui generis*, caused by the bacilli. WERNER.

COPPEY (249) observed a filaria in the anterior chamber of a negro infant, which six weeks before had come from Congo, and

at that time showed two small nodules on the iris, which soon disappeared. A thread worm one half *mm* thick and three *cm* long was seen moving in the anterior chamber. The eye was injected, the aqueous was clear, but the pupil could not be illuminated. The anterior chamber is the rarest location in the eye for the filaria, and to find a living specimen there is exceedingly rare. In most of the sixty cases in which Firket examined the blood of inhabitants of Congo, one or two filaria embryos were found in every drop of blood, so that it seems probable that in this case the filaria has been carried into the eye by the blood current.

Sections XII.–XXII. Reviewed by DR. SILEX.

XII.—IRIS.

250. WICHERKIEWICZ. Pseudo-gumma of the iris of traumatic origin. *Zehender's klin. Monatsbl.*, xxxii., p. 278.

251. FOURIER. Syphilitic iritis. *L'union méd.*, March 1, 1894.

252. MOCHECK. On miliary tuberculosis of the iris. *Wien. med. Wochenschr.*, No. 24–25, 1894.

253. SANDFORD. Three cases of tubercle of the iris. *Trans. Ophth. Soc. Unit. King.*, xiv., p. 90.

254. FERGUS. Note on peculiar ophthalmic conditions occurring in members of one family. *Ibid.*, xiv., p. 206.

WICHERKIWIECZ (250) reports two cases in which a foreign body had produced the clinical picture of gumma of the iris, and he remarks that we have no sure criterion of gumma, tubercle, and granuloma. On the other hand, in the case of a foreign body, an existing syphilis may be overlooked. A case of the latter sort was cured by specific treatment.

MOCHECK (252) observed three cases of tuberculosis of the iris in children. The two first died of tubercular meningitis. This was diagnosed in the third case also, but two years later the child was quite well. The features of miliary tuberculosis of the iris are as follows: The disease begins as a chronic iritis; there is little photophobia and the pain is slight. The moderate plastic exudation which usually blocks the pupil forms undetachable posterior synechiæ. There is always precipitation on the posterior surface of the cornea. The swelling of the

tissue is irregular, and the new formations are richly vascular. In the later course of the disease the miliary tubercles are usually visible.

SANDFORD'S (253) first case was one of miliary tubercles of the iris in a phthisical lad aged sixteen, and ended in recovery. The second case was exactly similar, occurring in a phthisical girl aged nineteen. The third case was one of rapidly growing solitary tubercle in a child aged five with very tubercular antecedents. As it was regarded as a case of primary tuberculosis of the iris, the eye was enucleated. The microscope revealed a tubercular structure, but no bacilli were found. Hill Griffith justly pointed out that it was impossible to distinguish between primary and secondary tubercle of the iris. WERNER.

FERGUS (254) relates two cases of corectopia and one of small dislocated lens in one family. WERNER.

XIII.—CHOROID.

255. WOLKOWITSCH. Exenteration of the eyeball. *Mittheilungen aus klinischen und medicinischen Instituten der Schweiz.* From the eye clinic at Berne. Basle and Leipsic, 1894.

256. COLLINS, E. T. Case of a primary tumor of the ciliary body, of glandular structure. *Trans. Ophth. Soc. Un. K.*, xiv., p. 83.

WOLKOWITSCH (255) calls attention to the cases of fatal meningitis following enucleation, and suggests evisceration as the best means of avoiding this complication. 750 cases, 154 of them from the Berne clinic, have been reported without a fatal outcome. The indications for the operation in the Berne clinic are: 1, panophthalmitis of exogenous or endogenous nature; 2, the more plastic and less purulent forms of acute, subacute, and chronic irido-cyclitis that follow injury and generally lead to loss of function in the one eye and danger to the second. Recovery takes place in eleven days on an average. Neurotomy is only made in painful absolute glaucoma in Berne. He concludes with an accurate description of the technique and the antiseptic precautions observed.

COLLINS (256) refers to similar cases published by himself (vol. xi.), and by Alt, Michel, and Lagrange. In the present case, a woman aged twenty-eight, there was no evidence of disease in any other part of the body. The pupil was occluded from iritis. T+1. The tumor was situated in the upper part of the ciliary region, extending from the root of the iris to the equator. The

anterior part was black, the posterior white. The growth presented a glandular appearance, with sarcomatous-looking collections of cells in the pigmented portion. WERNER.

XIV.—GLAUCOMA.

257. KUGEL. A case of return of qualitative perception of light after iridectomy in an eye amaurotic from glaucoma. *Graefe's Archiv*, xl., 3, p. 299.

258. SMITH, PRIESTLEY. An instance of hereditary glaucoma, and its cause. *Ophth. Review*, xiii., p. 215.

In KUGEL'S (257) patient, blind for three weeks, a double iridectomy was done in each eye at an interval of two weeks, and the patient was able to count fingers in the right eye at four feet. The deep excavation disappeared, and the vessels which had emerged at the margin of the disc again appeared in the centre.

PRIESTLEY SMITH (258) records cases of glaucoma in both eyes in a father and daughter, the latter aged twenty-nine. The eyes had been lost from absolute glaucoma with the exception of one of the daughter's eyes, in which vision was improved from motion of hand to $\frac{6}{24}$, by scleral puncture, followed by iridectomy. There was H = 6 D. The cornea measured 10.5 *mm* in the horizontal diameter in the father's eyes, and 10 *mm* in the daughter's eyes. The right eye of the daughter was excised for absolute glaucoma, and its dimensions were found to be subnormal, although the lens was slightly larger than normal. The filtration angle was closed. Small eyes generally contain disproportionately large lenses, due, according to the author, to the fact that the lens, in its capsule, has no vascular connection with the surrounding parts and is to a certain extent independent in its growth. Hereditary glaucoma would therefore seem to be caused by hereditary smallness of the eyeballs. Small eyes are attacked by glaucoma earlier in life than eyes of normal size; moreover, the recorded cases of hereditary glaucoma occurred at an early age.

Primary glaucoma seems to depend on various causes: sometimes on systemic, vascular, or nerve disturbance; sometimes chiefly on individual peculiarity in the build of the eye; and often on a complex co-operation of several causes. The author speaks favorably of scleral puncture as an adjunct to iridectomy, after several years of experience. WERNER.

XV.—SYMPATHETIC OPHTHALMIA.

259. ROHMER. Resection of the optic nerve according to de Wecker in sympathic ophthalmia. *Ann. d'Ocul.*, cxii., p. 249.

260. BRONNER. Case of sympathetic ophthalmia after concussion of the eyeball, with no visible external wound. *Trans. Ophth. Soc. Un. K.*, xiv., p. 215.

ROHMER (259) saw sympathetic inflammation follow resection of the optic nerve, making enucleation of the first eye necessary and permanently damaging the sight of the second. He favors enucleation solely in these cases.

BRONNER'S (260) patient developed sympathetic ophthalmia with K. punctata, eighteen days after a kick on the other eye. In the injured eye three was a small scleral staphyloma but no conjunctival wound, tension was never lowered, and inflammation was diminishing when the left eye became affected. WERNER.

XVI.—VITREOUS.

261. DIMMER. A case of hemorrhage between the retina and the vitreous. *Beiträge zur Augenheilk.*, xv.

262. BENSON. A case of "monocular asteroid hyalitis." *Trans. Ophth. Soc. Un. K.*, xiv., p. 101.

DIMMER (261) reports a case in which a large hemorrhage occurred between the retina and vitreous and partially covering the disc in a patient with albuminuric retinitis.

BENSON'S (262) patient, a gentleman, aged sixty-two, complained of slight asthenopia and muscæ. V = $\frac{6}{6}$ with each eye. Both eyes were normal with the exception of the vitreous humor in the right eye, which was studded with small, smooth spheres, like stars in the sky; they did not sparkle like cholesterine crystals. They were fixed, and by oblique illumination looked cream colored. No change occurred during nine months' observation. WERNER.

XVII.—LENS.

263. DALGANOFF. On the corneal astigmatism after extraction of cataract. *Wjest. Ophth.*, 1894, July-October.

264. WALKOFF. Is capsulotomy necessary in the extraction of senile cataract? *Ibid.*

265. WICHERKIEWICZ. The after-treatment of cataract patients after infection has occurred. *Resport of the Society of Natural Scientists and Physicians*, Vienna, 1894.

266. CZERMAK. On pressure bandages and rupture of the wound after extraction. *Wiener klin. Wochenschr.*, No. 27, p. 506.

267. THOMPSON, L. L. Observations on some phases of opacity, and on luxation of the crystalline lens. *Ophth. Review*, xiii., p. 313.

268. SNELL. Case presenting unusual appearances after extraction of cataract; simulating a cyst, really due to a distended capsule. *Trans. Ophth. Soc. Un. K.*, xiv., p. 135.

WALKOFF (264) gives a detailed report of 303 cataract operations done on 181 patients between 1891 and 1893, according to his method, which is as follows. The section is made in the cornea, the highest point coming a little above the upper margin of the pupil. The lens with its capsule is expelled by simultaneous pressure above and below with two spatulas. No iridectomy is done. The advantages of this operation are a round pupil, high acuteness of vision and simplicity of execution. The bandage is not removed for a week or longer. Complications and unfavorable results are not more frequent than with other methods of operation. In 22 cases the cataract was immature but the results were good in all. In 48 cases the capsule ruptured, but in 36 of these it was removed with forceps. There was loss of vitreous in 96 cases, in one of which the lens was not extracted. In 2 cases there was severe hemorrhage leading to atrophy. Traumatic simple iritis and irido-cyclitis occurred in 102 cases, in 49 of which there was prolapse or incarceration of the iris. Severe irido-cyclitis took place in 16 cases, in 4 of which division of the membranes gave vision of $\frac{8}{200}$ to $\frac{10}{200}$. Purulent keratitis and panophthalmitis occurred in 8 cases; incarceration of the iris in 72. V = o in 16 cases, in 10 of which the blame rested on the patient. HIRSCHMANN.

In beginning infection after extraction, WICHERKIEWICZ (265) advises sterilizing the margins of the wound with the galvano-cautery, 4–10 % nitrate of silver, or bichloride. If there is already exudation in the anterior chamber, the wound should be opened and the chamber washed out with physiological salt solution. Against infection of the vitreous we are powerless. For the inflammation, mydriatics should first be used, with cold applications in the early stages, and warm ones later.

CZERMAK (266) believes that prolapse of iris is best prevented by making a large section in the sclero-corneal margin. A pressure bandage favors rupture of the wound. For this reason he has discarded the bandage, and uses Fuchs's double shield.

THOMPSON (267) describes: 1. Segmental opacities of the lower

and inner portion of the lens. They are generally non-progressive, and occur in both eyes. The youngest patient was aged forty. 2. Annular opacities, or "arcus senilis lentis," the youngest patient being twenty-three. This form of cataract produces a spotted opacity of the anterior part of the lens after middle age. All the patients were women. 3. A case of fluid degeneration of cataract with restoration of vision. 4. Ectopia lentis. Four cases in relatives; all were highly myopic, with partial or complete iridodenesis, and the lenses were displaced outwards. Luxation of senile cataracts downwards is also referred to. WERNER.

In SNELL'S (268) case the lens was removed with vectis, with little loss of vitreous. V = $\frac{6}{9}$. Six or seven years later patient noticed a small white body in the ant. ch. There was a pear-shaped cyst at the lower part, attached to the iris by a slender filament, and was discovered to be the capsule of the lens, after removal. WERNER.

XVIII.—RETINA AND FUNCTIONAL DISTURBANCES.

269. POSSAUER. On the duration of life after the appearance of albuminuric retinitis. *Beiträge zur Augenheilk.*, xv.

270. DIMMER. The macular changes in retinitis albuminurica. *Prager med. Wochenschr.*, No. 42, 1894.

271. HUSEMANN. On tobacco amaurosis. *Deutsche med. Wochenschr.*, No. 43, 1894.

272. PETERS. On the occurrence and significance of the so-called displacement type of visual field. *Deutsche Zeitschr. f. Nervenheilk.*, v., p. 303.

273. KUNN. A case of embolism of a branch of the central artery of the retina, with remarks on the course of the macular arteries. *Wiener med. Wochenschr.*, No. 35, 1894.

274. LOMBARD. On a case of transient amblyopia following lead poisoning. *Rev. méd. de la Suisse Romande*, March 20, 1894.

275. WINTERSTEINER. On the structure, development, and genesis of glioma. *Wiener klin. Wochenschr.*, No. 27, p. 493.

276. BATTEN and SPICER. Congenital pigmentary plaques of retina. *Trans. Ophth. Soc. Un. K.*, xiv., p. 127.

277. BRAILEY. Notes of a case of failure of central vision. *Ibid.*, xiv., p. 123.

278. SPICER. Case of retinitis circinata. *Ibid.*, xiv., p. 132.

279. GRIFFITH. On a rare form of primary intra-ocular melanoma. *Ibid.*, xiv., p. 160.

280. COLLINS, E. T. Two cases, brother and sister, with peculiar vascular new growth, probably primarily retinal, affecting both eyes. *Ibid.*, xiv., p. 141.

The investigations of the Baroness POSSAUER (269) include 131 cases of albuminuric retinitis in chronic nephritis. It is not known exactly how often retinitis occurs in Bright's disease, the figures varying between 6 and 33 %. Many patients die soon after the appearance of the retinitis, but there were 5 cases in which the albuminuric retinitis was cured permanently. Among the Zurich patients the mortality of the men was greater than that of the women. Five women were living more than two years after the first ophthalmoscopic examination, and one eleven years after.

In eyes which showed ophthalmoscopically the macular star figure of retinitis albuminurica, DIMMER (270) found only collections of fatty degenerated cells in the outer nuclear layer of the retina. The stellate arrangement of the patches is due to the radiating arrangement of the cone fibres in the outer nuclear layer. HERRNHEISER.

HUSEMANN (271) gives a short report on epizoötic blindness in horses in Australia. The eyes are not inflamed, as in the forms previously known, but the disease leads to complete blindness in a year or two if the animal is not removed from the district. The disease is due to the Australian tobacco, *Nicotiana suaveolens*, and consists in an atrophy of the optic nerves.

PETERS (272) gives a *résumé* of previous articles on the displacement type of the limits of the visual field, and reports the findings in 150 patients examined by himself. In 98 cases it was wanting. Of the 52 cases showing it, 19 had nervous disturbances, while the rest were injured but had no nervous affection. In traumatic neurosis the symptom has no significance, being found in about half the cases. In 104 healthy individuals, the type was found in 23 % of cases.

In KUNN'S (273) case there was an embolism of the superior temporal branch of the central artery of the retina in a student, aged sixteen, with mitral insufficiency. There was a scotoma and normal central vision, due to the fact that the macula is mostly supplied by the small macular arteries which are sometimes given off low down, and which may then continue to supply the macula even when the main artery is blocked above them.

LOMBARD's (274) patient was a young man with lead-poisoning, who had an attack of colic followed by complete blindness. The pupillary reflex was lost and the discs were pale. The following day he counted fingers at 12 feet, and two days later the vision was so far restored that he could go out. SULZER.

Vision (276) was normal. No abnormality of fundus, except the plaques which were three or four in number, two being large; they are almond-shaped, with the pointed end directed towards the disc. They are red-gray in color, and not raised or depressed. The red reflex appears to shine through them, and the retinal vessels pass over them. WERNER.

BRAILEY's (277) patient, a man, aged fifty-seven, had slight pigmentary changes at the macula, with an area of superficial choroidal atrophy around the disc and m. l. Some of the choroidal vessels had thickened coats. WERNER.

SPICER's (278) patient, a woman, aged seventy-three, presented in the left eye the typical appearance of retinitis circinata, as described by Fuchs, without any trace of hemorrhage. In the other eye there were numerous hemorrhages without any white spots. WERNER.

In GRIFFITH's (279) case the melanotic growth sprang from the retinal pigment epithelium near the ciliary region; it was composed of columns and tubules of epithelial cells. After enucleation the growth recurred in the orbit and superior maxilla, and the patient, a man, aged thirty-seven, died within a year. WERNER.

In COLLINS's (280) first case the man's sight began to fail at age of twenty. There was detachment of the retina with enormous dilatation of vessels and development of white dots on its surface. Subsequently there was increased vascularity of the iris with glaucoma. The eye was enucleated, and the retina showed extensive changes, containing in one part a close plexus of small vessels. The sister became blind at twelve years of age, both eyes were staphylomatous, cornea opaque, and a dark bulging existed at the outer canthus of one eye. Microscopically both eyes contained bony plates in the choroid, no lens, iris, or ciliary body visible. A large mass involving the cornea in one eye existed in the globes, composed of small branching capillary vessels, having between them honeycombed cells with large nuclei. WERNER.

XIX.—OPTIC NERVE.

281. ELSCHNIG. On choked disc. *Report of the Society of German Natural Scientists and Physicians in Vienna*, 1894.

282. MOLL. A case of recurrent retrobulbar neuritis. *Centralbl. f. prakt. Augenheilk.*, xviii., p. 269.

283. JULER. Uniocular optic neuritis. *Trans. Ophth. Soc. Unit. King.*, xiv., p. 121.

284. SANDFORD. Case of double optic neuritis from caries of sphenoidal cells and intracranial abscess. *Ibid.*, p. 119.

285. WILLIAMS, R. A case of double optic neuritis. *Ibid.*, p. 118.

286. TAYLOR. Optic neuritis in its relation to intracranial tumor and trephining. *Ibid.*, p. 105.

ELSCHNIG (281) calls those cases choked disc only when the disc projects 2 D. The examination of 55 cases showed an inflammation of the disc in so far as the discovery of increase in nuclei and infiltration of the central connective-tissue tract. The swelling is due to œdema, which never affects the lamina cribrosa. The optic nerve always shows changes in the form of interstitial neuritis and perineuritis. Hydrops of the optic-nerve sheath was not constant. In 21 cases of brain tumor inflammation in the meninges was found 13 times.

MOLL'S (282) case was that of a young man in whom a retrobulbar retinitis developed in both eyes, and recurred three times in four years. The function remained good, while the discs showed white atrophy. The cause was probably a chronic periostitis about the optic foramen.

JULER'S (283) patient, a woman aged twenty-eight, had syphilis; hemicrania was the only other symptom beside the loss of vision. No P L. WERNER.

The only observable symptoms in SANDFORD'S (284) case, a man aged seventy-eight, were blindness and optic neuritis two years and a half before death, which took place without paralysis or previous loss of consciousness. There was a large encysted abscess at the base, between the layers of dura mater in the middle fossa. The walls of the sphenoidal sinus were completely destroyed. WERNER.

WILLIAMS'S (285) patient, a woman aged sixty-five, suddenly lost her sight, without any symptoms, headache or otherwise. There was slight haziness of the discs, pupils dilated and inactive.

She was given potass. iodid., and in six weeks V = $\frac{6}{18}$. Fields normal. Neuritis gone. WERNER.

TAYLOR (286) records nine cases of trephining for cerebral tumor, in which optic neuritis was present. In the first three cases the tumor was removed and the optic neuritis subsided; in one of these the discs were normal four months after operation, and in another the tumor recurred, but only slight optic neuritis reappeared in the final stage. In the second group twice the tumors were seen, but not removed, and in a third no growth could be found, nevertheless the neuritis subsided, although in one case the tumor continued to grow. In the third group, the tumors were removed, but recurred in two cases without redevelopment of neuritis. A cyst was tapped in the last case, with subsidence of the neuritis, although the patient died of tumor. No active treatment by drugs was carried out after operation. These cases seem to favor the view that increased ntracranial pressure is the effective agent in producing optic neuritis. In the discussion, Mr. V. Horsley stated that there were a sufficient number of cases of recurrence of the growth without recurrence of the neuritis to show that even if the dura mater be not opened, the optic neuritis disappears. WERNER.

XX.—INJURIES, FOREIGN BODIES (PARASITES).

287. SCHIRMER. The Mackeown-Hirschberg method of magnet extraction. (An answer to Prof. Hirschberg.) *Deutsche med. Wochenschr.*, No. 29, 1894.

288. PERLES. On the removal of foreign bodies from the eye. *Berl. klin. Wochenschr.*, No. 28, 1894.

289. HIRSCHBERG. On the removal of iron splinters from the retina. *Deutsche med. Wochenschr.*, Nos. 25 and 26, 1894.

290. ARGYLL-ROBERTSON. Case of trichosis bulbi. *Trans. Ophth. Soc. Unit. King.*, xiv., p. 196.

Since Haab's publication, many objections have been made to HIRSCHBERG'S (289) method of extracting foreign bodies from the eye, and some have thought that the results were not good. Hirschberg has done the operation 150 times, and has never seen suppuration in the wound, and suppuration in the interior but once. The careful introduction of a clean magnet is not dangerous. Loss of vitreous is rare, and bruising of the vitreous is avoided when a sharp knife is first passed through the vitreous in

the direction of the foreign body. Foreign bodies whose location is unknown may be removed with a small magnet. The bad results depend chiefly upon the severity of the injury. Sometimes the foreign bodies are so firmly imbedded that removal is impossible.

This (290) was a dermoid situated on the upper and outer part of the sclerotic under the upper lid ; a few large hairs grew from its surface. WERNER.

XXI.—OCULAR DISTURBANCES IN GENERAL AFFECTIONS.

291. WESTHOFF. Diabetic inflammations of the eye. *Medisch. Weekbl.*, p. 416, 1894.

292. PEL. Hemianopsia. *Ibid.*, p. 407.

293. ANDOGSKY. On eye inflammations from intestinal worms. *Zehender's klin. Monatsbl.*, xxxii., p. 263.

294. BERGMEISTER. On ocular disturbances with certain anomalies of metabolism, particularly the increase of uric acid. *Report of the Society of Natural Scientists and Physicians in Vienna*, 1894.

295. BOGMANN. Contributions to the study of ocular affections during lactation. *Ann. d'Ocul.*, cxii., p. 161.

296. FREYHAN. A case of tubercular meninigitis that recovered. *Deutsche med. Wochenschr.*, No. 36, p. 707.

297. KNAPP. A case of otitic brain abscess successfully operated. *Arch. of Otol.*, xxiv., p. 155.

298. AXENFELD. On purulent metastatic ophthalmia, its etiology and prognosis. *Grafe's Archiv*, xl., 3 and 4.

299. BENSON. A case of recurrent transitory blindness. Eighth International Congress. *Arch. f. Augenh.*, xxix., 3–4, p. 378.

300. DEJERINE and VIALET. On a special form of functional hemianopsia in neurasthenia and traumatic neurosis. *Ann. d'Ocul.*, cxii., p. 147.

301. STERN. On periodic variations in the functions of the cerebral cortex. *Berl. klin. Wochenschr.*, 1894, p. iiii.

302. SEREVRENNIKOWA. A case of amaurosis due to gumma of the base of the brain. *Wjest. Ophth.*, 1894, June–October.

303. IWASA. On a case of tubercle in the optic thalamus. *Inaug. Dissert.*, Freiburg, 1894.

304. WERNICKE. Complete right homonymous hemiopia after a perforation wound in the left occipital region. *Deutsche militärärztliche Zeitschr.*, 1894, part ii.

305. JACOB. On a case of hemiplegia and hemianæsthesia and crossed oculomotor paralysis with unilateral destruction of the optic thalamus, the posterior part of the internal capsule, and the anterior corpus quadrigeminum, with particular reference to the secondary degenerations. *Deutsche Zeitschr. f. Nervenheilk.*, x., pt. 2 and 3.

306. WILBRAND and SAENGER. On the visual disturbances in functional nervous affections. Leipsic, 1892.

307. GUIBERT. Poliencephalitis superior and inferior. Recovery. *Arch. d'opht.*, xiv., 9, p. 543.

308. LONG and BEEVOR. Binasal hemiopia in a case of tabes dorsalis. *Trans. Ophth. Soc. U. K.*, xiv., p. 246.

WESTHOFF (291) describes a case similar to Horner's, in which the refraction changed in a diabetic patient. A man of fifty-one stated that four days previously he suddenly found that he saw poorly near by and clearly in the distance. Previously he had worn — 1.D for distance and read without glasses. The patient was found to have hyperopia in one eye and hyperopic astigmatism in the other. Fourteen days later the patient had myopia again and read without glasses.

PEL (292) demonstrated a patient aged forty-two, who suddenly noticed that he could not see the right half of his paper. There was also disturbance of intellect and speech, and paralysis of the right leg and face. The hemianopia was permanent, the other symptoms dissappeared. WESTHOFF.

According to ANDOGSKY (293) a great number of the eye diseases observed in children with intestinal worms depend upon the disturbances in the formation and circulation of the blood thus caused. The disturbances are usually functional only, but there may be neuro-retinitis and hemorrhages in the retina. Reflexly there may occur mydriasis, myosis, spasm of accommodation, strabismus, and blepharospasm. Andogsky describes a unilateral blepharospasm in a girl of fifteen and a bilateral blepharospasm in a girl of eleven, which resisted every form of treatment, but disappeared at once after the removal of the worms.

In 4 cases with increase of uric acid in the urine, BERGMEISTER (294) found posterior polar opacities of the lens, which disappeared as the uric acid grew less. He also found retinal hemorrhages and neuro-retinitis.

BOGMANN (295) sketches the history of visual disturbances during lactation from their earliest description to the present. His own patient had a double optic neuritis coming on in the third week after parturition following the sudden cessation of the secretion of milk. SULZER.

FREYHAN'S (296) patient, a man of twenty, had the usual clinical symptoms of tuberculous meningitis, the diagnosis being confirmed by the discovery of tubercle bacilli in fluid obtained by puncture. Ophthalmoscopically there was neuro-retinitis. The case recovered completely.

In a girl aged nine, KNAPP (297) found chronic otorrhœa, headache, fever, choked disc, and homonymous hemiopia. On account of the general symptoms and the absence of other nerve affections, an otitic cerebral abscess was diagnosed and a radical mastoid operation and trephining were done. An abscess in the temporal lobe was evacuated. A cerebral hernia resulted. The general condition was bad until two small superficial abscesses were evacuated, when the hernia retracted and the child recovered.

AXENFELD (298), for the purpose of determining the prognostic significance of metastatic ophthalmia, reports 14 cases of his own examined clinically and bacteriologically, and reviews the 200 cases in the literature.

His conclusions are as follows: Purulent metastatic ophthalmia is complicated with ulcerative endocarditis in one third of the cases. To this, with the frequent degeneration of thrombi is due the frequency of the diseases after parturition. In one third of the cases it is bilateral and when following parturition is almost always fatal. Unilateral metastatic ophthalmia often accompanies slight attacks of pyæmia, particularly the cases without assignable cause, and those following affections of the lungs or meninges. It is mostly an affection of the capillary vessels of the retina, as is shown by the fact that, although blindness comes on rapidly, it never occurs suddenly, as it would if larger vessels were stopped. Often the ocular metastasis is the only one found.

In the puerperal processes the cause is the streptococcus in most cases, in surgical cases this and the staphylococci, these micro-organisms usually causing panophthalmitis. The Fränkel-Weichselbaum pneumococcus is the cause of some cases and these usually end in phthisis without panophthalmitis.

According to the observations of DEJERINE and VIALET (300) concentric contraction of the visual field is not the only form of limitation of the field found in neurasthenia and traumatic neu-

rosis. Both affections can cause hemiopia, which, in spite of long duration, is only functional and has no anatomical foundation. It differs from ordinary hemiopia in the marked variations of the limits of the preserved half of the field. In two patients, one with extreme spontaneous neurasthemia and one with traumatic hysteria, the authors had observed the type of hemiopia described, during four years. SUĹZER.

The new variety of nervous functional disturbance described by STERN (301) consists in a periodic decrease in the function of the cerebral cortex. There is an intermittent decrease in the sensibility of all the sensory regions, a paresis with ataxy of the voluntary muscles, and a decrease in intellectual power. In one case the vision, usually $\frac{6}{5}$, decreased to $\frac{1}{15}$, with simultaneous contraction of the field.

SEREVRENNIKOWA'S (302) patient, a man of twenty, five months after the chancre for which he had received mixed treatment, developed severe headache, which, in spite of treatment, was soon followed by loss of sight in the right eye. In two weeks the left eye was completely blind and the right had begun to fail, and was blind ten days later. Following this there was paralysis of both oculomotor nerves. Ophthalmoscopically nothing abnormal was found. The sense of smell was weakened, and the forehead and scalp were sensitive to pressure. A diagnosis of gumma behind the chiasm was made. At the autopsy, eight months after infection, there was found a gumma as large as a walnut in the cerebral substance, behind the chiasm, above the left optic tract, and a smaller gumma more to the right. HIRSCHMANN.

WERNICKE'S (304) case was peculiar from the fact that the hemiopia was the only symptom of the cerebral injury. The first occipital convolution must have been affected. The transitory ptosis was thought to be due to the influence exerted from a distance on the gyrus angularis which lies anterior and is probably the cortical centre for the extrinsic ocular muscles.

GUIBERT'S (307) case was a woman of twenty-five in whom within a short time severe headache developed and a paralysis, apparently bulbar, of all the ocular muscles on each side. Vision was reduced to $\frac{1}{4}$, field normal, and fundus normal. The urine contained sugar. Although the case did not appear to be syphilitic, it must be considered such, as it was completely cured by the injection of oleum hydrarg. biniodid. and the internal use of potas. iodid. followed by galvanization.

In this case (308) there was atrophy of discs. V $\frac{6}{24}$ and $\frac{5}{60}$. Red-green blindness. WERNER.

MISCELLANEOUS NOTES.

APPOINTMENTS.

Prof. I. SCHNABEL began his functions as director of the Second Eye Clinic in Vienna, April 3, 1895.

Prof. W. CZERMAK, of Innsbruck, has been appointed director of the Eye Clinic in the German Medical Faculty of Prague.

MEETINGS OF SOCIETIES.

BRITISH MEDICAL ASSOCIATION, in London, July 30th to August 2d, inclusive.

GERMAN OPHTHALMOLOGICAL SOCIETY, 4th to 7th August, at Heidelberg.

At the last meeting of the GEORGIA STATE MEDICAL ASSOCIATION an OPHTHALMOLOGICAL SECTION was organized. Dr. J. L. Hiers, of Savannah, was elected President, and W. F. Aiken, of Savannah, Secretary. Other members enrolled were: Calhoun, Hobbs, and Ray, of Atlanta; Hull and Tinsley, of Augusta; Shorter, of Macon; Paige, of Savannah.

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X. THE SAME. Three unusual Cases of Retino-Choroidal Degeneration. Plate III. (To appear in our next number.)

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XVI. S. HOLTH. Light, our Yellow Ointment, and the Salve-Jars in common use.

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XIX. A. DARIER. Subconjunctival Injections. Reply to Drs. Mellinger and Gutmann.

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XXI. S. LACHOWITZ. The Bacteria of the Healthy Human Conjunctiva.

XXII. C. SCHWEIGGER. Note on the Mechanism of Accommodation.

B.—REPORT ON THE PROGRESS OF OPHTHALMOLOGY IN THE THIRD QUARTER OF THE YEAR 1894. (Translated in this number.)

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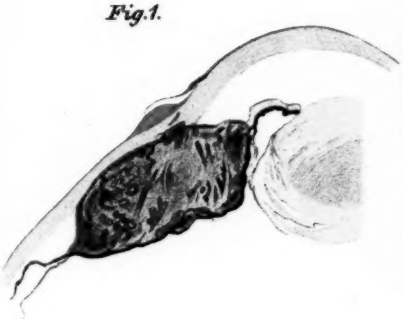

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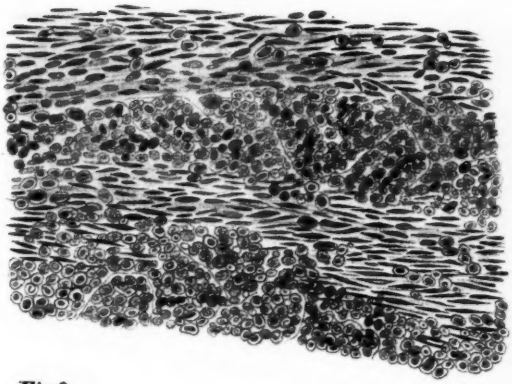

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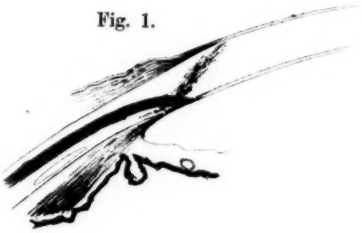

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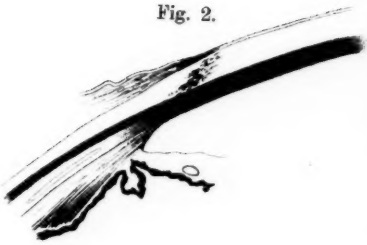

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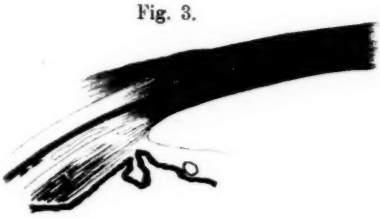

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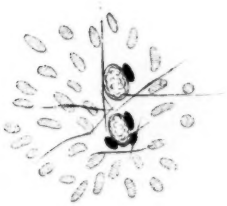

Fig. 7.

VERLAG VON J. F. BERGMANN, WIESBADEN.

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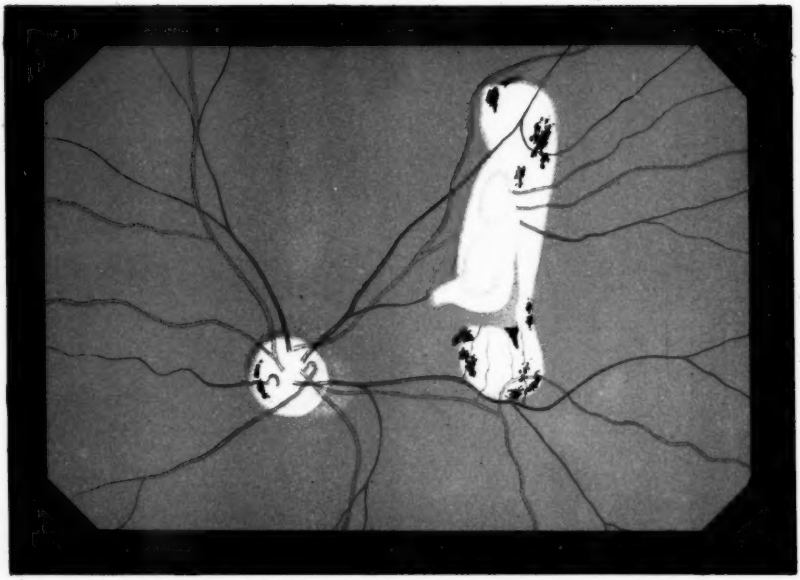

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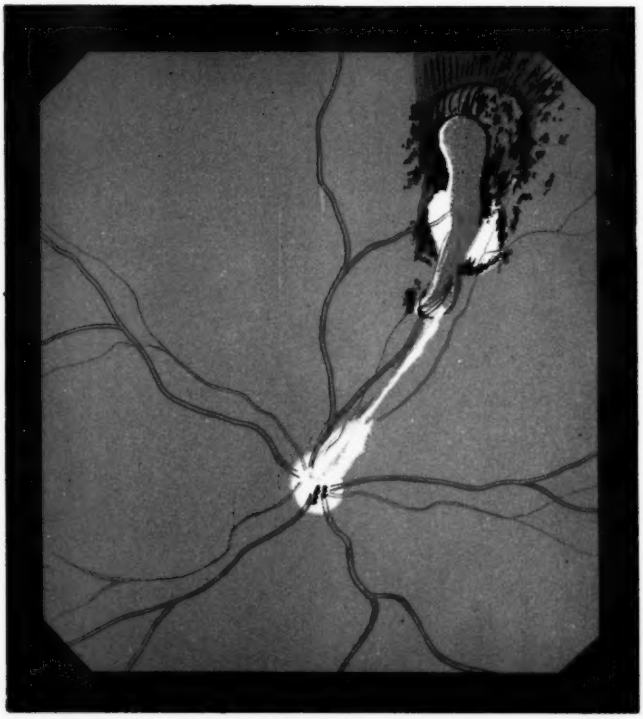

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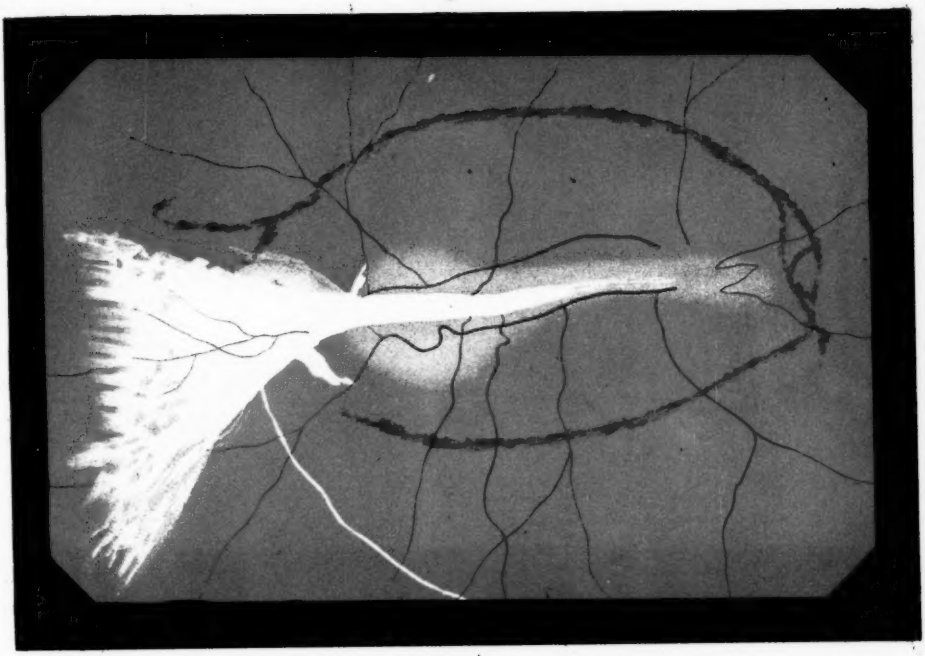
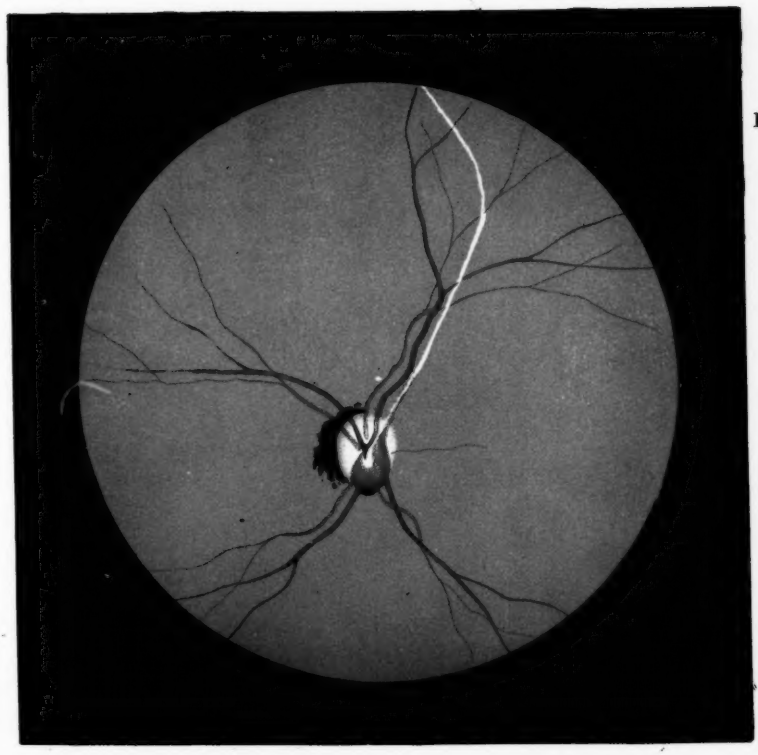
Fig. 3.





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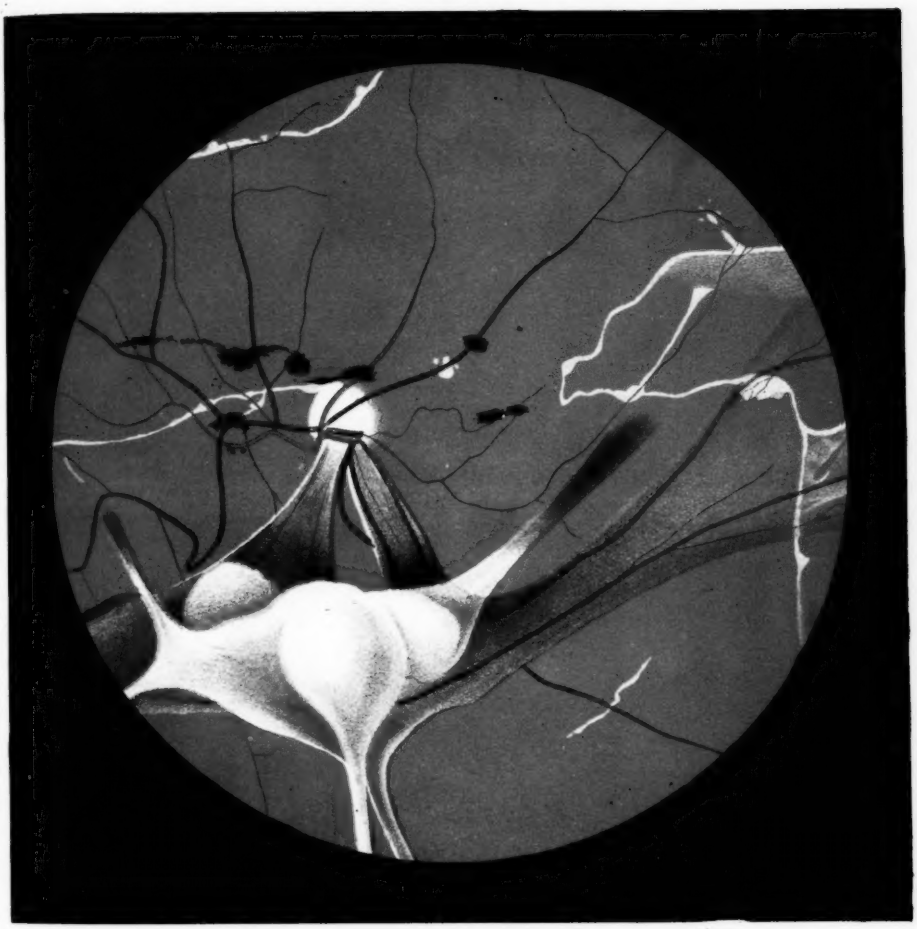

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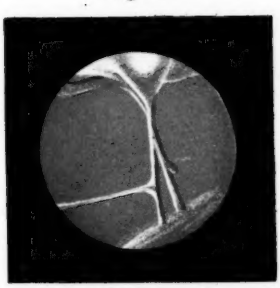

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ARCHIVES OF OPHTHALMOLOGY

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The ARCHIVES OF OPHTHALMOLOGY contain *original papers* on all branches of ophthalmic surgery, and *original reports* on the progress of ophthalmology. The original papers occupy from two thirds to three fourths of the space, and their scope embraces all subjects of scientific and practical interest connected with this department of medicine.

The object of the ARCHIVES is to guard, solidify, extend, and diffuse the stock of acquired ophthalmic knowledge by recording the constant addition of new observations, investigations, methods and means of examination, clinical experience, instruments and remedies, and further by taking part in the discussion of the questions of the day with a spirit alike conservative and progressive.

Particular attention is paid to the preparation of the Report on the Progress of Ophthalmology. The report is intended to furnish a *complete, systematic, and early* review of the current ophthalmological literature of the world. It has been necessary to divide the work of its preparation among a number of collaborators according to the following arrangement:

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1. *General Ophthalmological Literature:*
   a.—Text-books (not abstracts, but notes pointing out what is new and characteristic).
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   c.—Periodicals on our specialty (short notices of their character, time and place of publication, the titles of the original papers, with the names of their authors).

2. *General Pathology, Diagnosis, and Therapeutics*, including new instruments, apparatus, and remedies.
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3. *Lachrymal Apparatus.*
4. *Muscles and Nerves.*
5. *Orbit and Adjacent Cavities.*
6. *Conjunctiva, Cornea, Sclerotic.*
7. *Injuries and Foreign Bodies of these parts.*

Dr. P. SILEX, Lecturer at the University of Berlin.

1. *Uveal Tract*, including glaucoma.
2. *Refracting Media*, including the lens.
3. *Retina and Optic Nerve.*
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For Germany: Drs. BERNHEIMER, Vienna, HORSTMANN, SILEX, and DEUS, in Berlin.

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Under the heading of "Miscellaneous Notes," there will be published all kinds of professional news which specially concern the oculist, *e. g.*, appointments, honors, resignations, vacancies, new ophthalmic hospitals, opportunities for instruction, prize questions and essays, announcement of Society meetings, etc. Brief notices of this kind will be thankfully received by the editors.

Original papers of value from any source will, as heretofore, be welcome to the ARCHIVES, and are solicited.

EDITORS:

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d. Words to be printed in *italics*, should be underscored once, in SMALL CAPITALS twice, in LARGE CAPITALS three times.

4. Authors may receive proofs for revision if they will kindly return them without delay. We beg however to remind our contributors that changes in the copy are equivalent to resetting, causing so much additional expense. We therefore request them, to make, if possible, no alterations at all in their MSS., or, at least, to limit these to what is of essential importance.

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